THE
LARYNGOSCOPE.

VOL. XLVII MAY, 1937. No. 5

THE DIAGNOSIS OF DISEASES OF THE NEURAL MECHANISM OF HEARING BY THE AID OF SOUNDS WELL ABOVE THRESHOLD (PRESIDENTIAL ADDRESS).*

DR. EDMUND PRINCE FOWLER, New York.

It seems appropriate to preface the clinical portion of this Symposium on the Neural Mechanism of Hearing by further observations on the phenomenon of the recruitment of loudness with intensity, which I first reported before the American Otological Society in 1928,[1] because last year I described a method for using it in differential diagnosis.[2] My address will serve to link the laboratory and experimental with the clinical divisions of the program.

The only way to determine in the living the presence, extent and type of auditory neural lesions is to test the hearing for sounds of various frequencies and intensities. There are many means for doing this, but every method yet devised is frequently uncertain and often misleading, even though its limitations are appreciated.

The tests most commonly relied upon for diagnosis and some of their shortcomings may be briefly stated as follows:

1. Total deafness. Even complete unilateral deafness may be undiscovered, because of failure to properly mask the opposite ear. Total deafness may be simulated.

*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 28, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 17, 1937.

2. Bone conduction lateralized to the better ear (the Weber test). This is often misleading, because it may be referred to one ear using one frequency, and to the opposite ear using another frequency; to one ear with high intensities and to the opposite ear with low intensities (see Fig. 1).

3. Lowered hearing for the high tones by air conduction (lowered upper tone limit). This symptom is not pathognomonic unless bone conduction is also lowered at the same frequencies, because obstructive lesions may also cause a high tone loss, as I have shown experimentally by loading the malleus with shot.[3]

Tinnitus is a frequent cause of loss of hearing for the high tones, and may be caused by other than neural lesions; but, no matter what the origin, it cuts down the hearing for both air conduction and bone conduction for tones of its own frequencies and for tones above it in frequency. It may be considered a form of nerve deafness.

The high tones of the Galton whistle and monochord are so loud that they may be heard in the presence of even marked deafness in either the ear being tested or in the opposite closed ear. They may appear even as loud to the deafened ear as to the normal ear.

4. Air conduction better than bone conduction (positive Rinné). Using audiometric methods and the same normal base line, this rarely, if ever, obtains, because the difference between air conduction and bone conduction is the measure of the impedance in the conduction mechanism of the ear. If there is no middle ear lesion, air conduction is the same as bone conduction. If there is an impedance lesion, air conduction will be less than bone conduction. Unless the opposite ear is masked it is often impossible to tell which ear is actually hearing the bone conduction sound, which ear is being measured, because the impedance across the skull is only 4 or 5 dcb. Masking is seldom carried out as a routine technique, which is one reason why monaural nerve deafness is not more frequently recognized.

5. Diminished bone conduction (shortened Schwabach). This is generally thought to be the most reliable test for nerve deafness. It seldom occurs without an equal or greater loss by air conduction, although it is conceivable that it may

do this with certain lesions involving the cochlea windows, which equalize the release of sound pressures in the two scalae, so that bone conduction is diminished without any neural lesion.

Bone conduction is often seemingly lowered because the testing room noises or the sounds used to mask the opposite ear are so loud that they also mask the ear being tested. Bone conduction lowered at one or two of the frequencies below 1,000 d.v. is not a reliable sign of nerve deafness, because it occurs in many instances when the higher frequencies are not lowered by bone conduction (see Fig. 1).

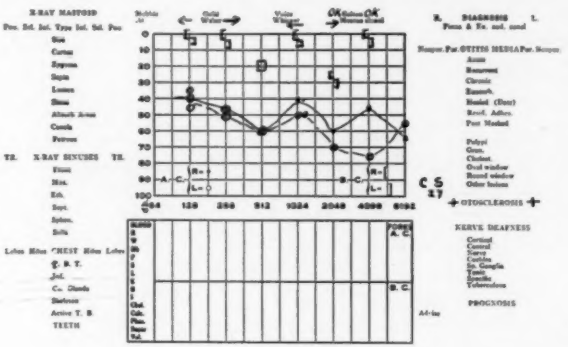

Fig. 1.

I call attention to the fact that any or all of the standard tests may appear positive and yet there may be no nerve deafness, that all are fundamentally dependent upon bone conduction for diagnostic significance and that bone conduction is often unreliable.

The method I described last year makes it possible to differentiate neural from conduction deafness without recourse to either air conduction or bone conduction threshold measurements and, except in certain instances, to be described anon, without using bone conduction in any form. It is the only reliable test for neural deafness which does not require confirmation by other tests. The test consists in measurement of the recruitment of loudness with intensity, by bal-

ancing the loudness of sounds, thrown alternately first into one ear and then into the other ("the alternate binaural loudness balance"). The varying differences in loudness necessary for balance at the various steps above threshold are the measures of the rates or recruitment of the neural elements necessary for equal balance. They are, therefore, a measure of the location, number and extent of loss of the neural elements which either initiate, conduct or relay impulses from the end-organs to the cortex.

It may be advisable to briefly restate the fundamental observations described in detail last year, namely:

1. In monaural (or unequal binaural) pure conduction deafness there is no change in the relative sensations of loudness

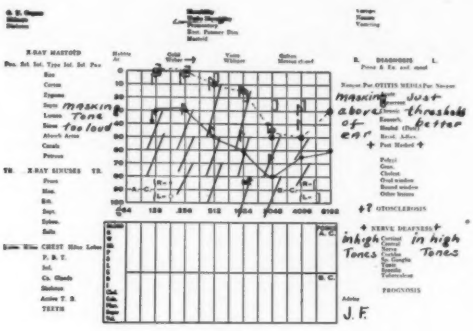

Fig. 2.

in the two ears at any intensity, *i.e.*, if there is a difference in hearing of say 30 dcb. at threshold, there will be a difference in the hearing of 30 dcb. for all intensities above threshold. In other words, it will take 30 dcb. (the threshold difference between the two ears in decibels) added to the poorer ear to make the sound appear as loud to it as to the better or normal ear.

2. In monaural (or unequal binaural) nerve deafness the poorer hearing ear hears more and more acutely as the intensity of the sound increases, so that, although there may be a difference of even as much as 75 dcb. between the hearing of the two ears near thresholds, there may be little or no

difference between the sensations of loudness in the two ears at some intensity well above the thresholds.

The phenomena described above occur in varying degrees at different frequencies, and vary with certain types of curves. I observed several apparent exceptions.

Fig. 2 illustrates an absence of recruitment of loudness in what appeared to be an ear with nerve deafness. The bone conduction almost coincided with the lowered A.C. curve. This warranted a diagnosis of nerve deafness, but on subsequent examination the masking sound was proved to be so loud that it masked the hearing in the ear being tested and thereby made it appear that bone conduction was down 20 to 40 dcb. more than it actually was. When I used a

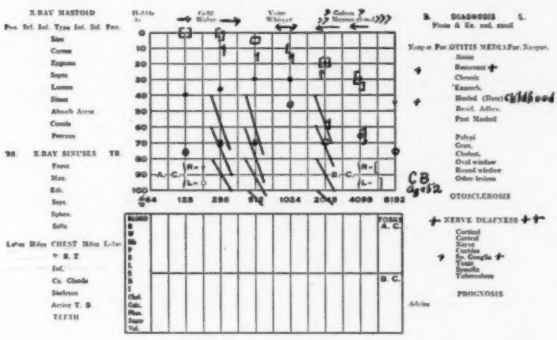

Fig. 3.

broad band of frequencies, just sufficient to mask the better ear, *i.e.* (only 40 dcb. above threshold for 256, 50 dcb. above for 512, 75 dcb. above for 2,048, and 70 dcb. above for 4,098), the hearing by B.C. was, with the slight differences at the higher frequencies, practically the same as for the better ear. The balance phenomenon had proven to be more dependable for diagnosis than the bone conduction tests. Several other similar exceptions have been similarly explained. This experience shows how important it is to avoid over-masking.

Fig. 3 illustrates the reverse of Fig. 2. The presence of recruitment of loudness in what appeared to be an ear with obstructive deafness. The B.C. in the better (near normal)

ear and in the worse ear appeared nearly the name (*i.e.*, near normal) and Weber was referred to the worse ear at least for the low tones, but the ears unexpectedly responded to the loudness balance by rapid recruitment in the left (worse) ear. Careful masking demonstrated a 10 to 15 dcb. loss by B.C. in the left ear at the lower frequencies and a marked loss at the higher frequencies. Here again the recruitment of loudness phenomena was a better diagnostic guide than the usual bone conduction tests.

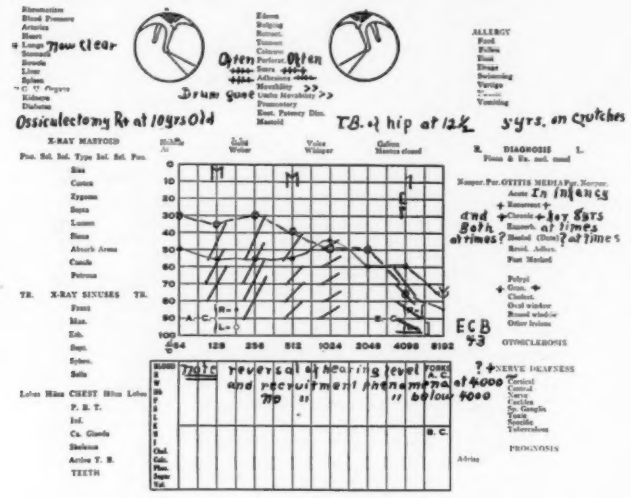

Fig. 4.

Note that Weber was referred to the worse ear using the lower frequencies and to the better ear using the higher frequencies.

Fig. 4 illustrates a reversal of form as between the low tones and the high tones. The left ear hears better than the right up to 1,000, but thereafter the right hears better than the left (at minimum audibility). Not so, however, for loud sounds, as is seen by the graph, because for the low tones the difference in hearing, being of conduction origin, shows no tendency to equalize as between the two ears; but for the higher tones there is a distinct leveling up of loudness val-

ues as between the two ears (they heard equally well at loud intensities). All tests indicate that there is a nerve deafness present in the better ear for the high tones. The loudness balance ran true to form at all frequencies.

Note: There is invariably a difference in the quality of the sound heard in the poorer (nerve deafened) ears. It is cloudy or less clear and often appears to be different in pitch. It appears at threshold suddenly and distinctly—there is no doubt about its being heard; in conduction deafness the reverse is true—sounds just above threshold appear and disappear, are indistinct and there is doubt as to whether they are heard or not, or often as to whether they are felt.

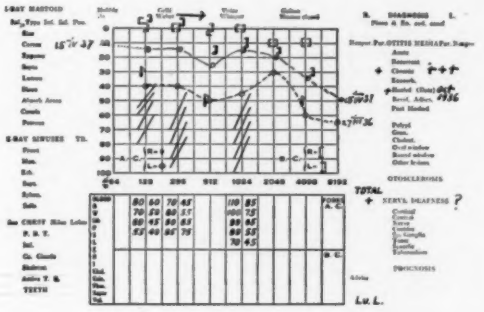

Fig. 5.

In cases of monaural total deafness, the loudness balance lines show no tendency to level up. They will, however, be longer the nearer the hearing ear is to normal (see Fig. 5, L.L.). In this case, when the hearing improved 20 dcb., the balance lines (which indicated the impedance across the head) showed an increase of 20 dcb., making the balance difference 40 dcb. instead of the prior 20 to 25 dcb. The B.C. decreased about 10 dcb., not because of any change in the neural mechanism, but because of lessened impedance in the middle ear.

At 8,000, and often at 4,000, it is difficult to obtain dependable B.C. measurements, because most receivers and all tuning forks permit the sounds to be heard by way of the external auditory meatus. The binaural balance test in such cases gives a more reliable measurement than the B.C. tests. More-

over, B.C. is more difficult to gauge than A.C. It may vary as much as 20 dcb. under apparently identical conditions. It may, therefore, appear near normal or even above normal in the presence of nerve deafness.

If the hearing curves are the same for both ears, it is still possible to use the balance test (see Fig. 6). Compare one or the other ear at a low frequency with one or the other ear at some higher frequency which shows a marked loss; *i.e.*, straddle the frequency interval by balancing the loudness as between the two chosen frequencies (as shown by Fletcher and Steinberg). If the lines joining the same loudness levels, at the two frequencies, tend to level up (to become more

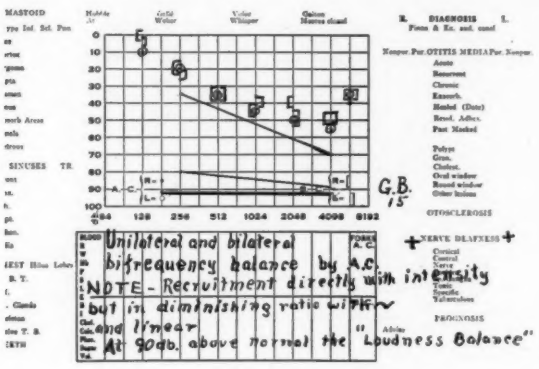

Fig. 6.

horizontal) as intensity is increased, it indicates the difference in the A.C. levels is due, at least in part, to a neural lesion affecting the higher tones.

If the air conduction graphs for both ears are the same and horizontal throughout, and B.C. is better than A.C., it is still possible to use the balance test by balancing the B.C. of one or both of the ears against the A.C. of one (or both) of the ears.

Fig. 7 shows monaural and binaural hi- and multi-frequency "Loudness Balance." The asterisks indicate points of equal loudness. All this proves that the recruitment of loudness runs true to form in all types of nerve deafness.

Fig. 8 (S.I.R.) illustrates in a case of monaural nerve deafness (neuroma of VIIIth nerve involving both divisions) the balance phenomena at three frequencies, 256, 1,024 and 4,096. The left (deaf) ear is down about 50 dcb. below the right (normal) ear at all these frequencies and the measurements are, therefore, more simple and understandable than in uneven audiograms. There is a progressive lag in the rate of recruitment of loudness with frequency. It required only 20 dcb. above minimum audibility at 256 for the left (deaf) ear to obtain the same loudness sensation as in the right ear after an increase of 65 dcb. In other words, the two ears balanced with equal intensities at 70 dcb. above normal

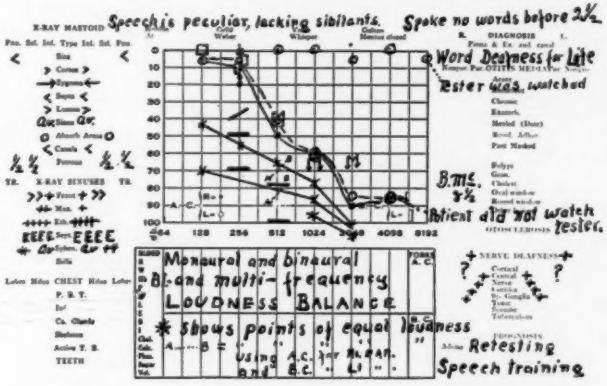

Fig. 7.

threshold (65 dcb. above the threshold of the right ear). There is present a conduction deafness of 15 dcb. in the left ear at this frequency.

At 1,024, it required an increase of 25 dcb. in the left ear to obtain the same loudness sensation as in the right ear after an increase of 80 dcb.; *i.e.*, the two ears balanced with equal intensities at 85 dcb. above normal threshold (80 dcb. above the threshold of the right ear). There is apparently no conduction deafness in the left ear at this frequency.

At 4,096, it required an increase of over 35 dcb. in the left ear to equalize the sensations with the same intensities.

There is 30 dcb. of conduction deafness in the left ear at this frequency.

In this case (and in all similar cases) a lag occurs in the recruitment of loudness, directly with frequency. The explanation is suggested that this lag is related to the fact that the higher the frequency the fewer the nerve elements available for recruitment.

I find that there is often a difference in the recruitment ratio with frequency, and that this varies with the type and amount of hearing loss in the two ears; in other words, with

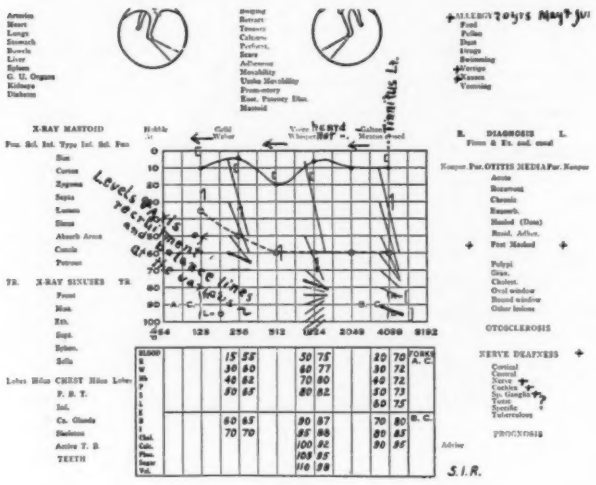

Fig. 8.

the location and extent of the neural lesion. The thought is that a spiral ganglion (or basilar membrane) scatter lesion should react differently than block lesions, or pure cochlear nerve lesions, or auditory nuclei lesions. Irrespective of whether the lesion be peripheral or central, if the ratio of the recruitment of loudness is high, the inference is that the cortical mechanism is intact, at least on one side.

In this patient the very loud shouted voice did not show balance at equal loud intensities; in other words, it always appeared somewhat louder in the normal ear than in the

opposite deafened ear. This phenomenon is, of course, the rule in impedance deafness. It has also been observed in certain cases of neural deafness. The explanation is that in the latter the balance intensity varies with frequency and, therefore, when using a broad band of frequencies, equal balance cannot be attained simultaneously for all frequencies at any intensity level.

In conclusion, I show (see Fig. 9) a series of graphs which clearly demonstrate how at any frequency the "alternate binaural loudness balance" measures the recruitment of loudness in deafness from various combinations of middle ear

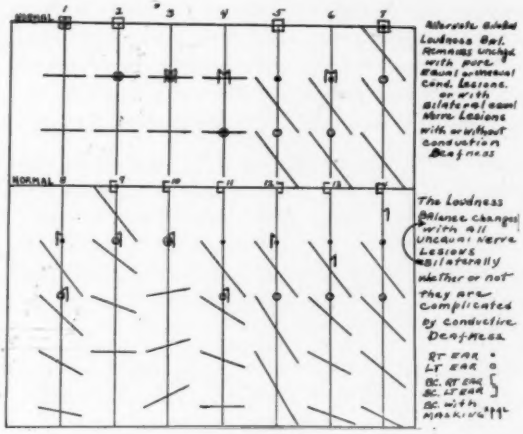

Fig. 9.

(impedance) and neural mechanism lesions. These graphs are all founded upon observations of actual cases.

Attention is called to the invariable recruitment phenomenon in every case where nerve deafness is indicated by a loss in bone conduction.

Attention is also called to the fact that at times the ear deafened from a nerve lesion may appear to hear even better than the opposite, better ear. The real significance of this phenomenon is obscure.

From the data set forth, it is apparent that it will require less intense masking noises to mask an ear with nerve deaf-

ness than one with impedance deafness and, therefore, that whenever an ear is easily masked (*i.e.*, by a sound at or just above its threshold), it is either a normal ear (which can be easily verified) or an ear suffering from a lesion of the neural mechanism of hearing. It follows that even a faint tinnitus at or below the testing frequency will readily mask an ear with neural deafness, and that the true threshold may be observed only in the absence of tinnitus. However, the ratio of loudness recruitment furnished a means for estimating the masking effect from tinnitus.

If tinnitus is of purely middle ear origin, the loudness balance shows a tendency to level up only until the tinnitus is masked by the testing frequency. After this, the loudness difference between the two ears is maintained throughout the rest of the intensity scale. If tinnitus is of purely neural origin, the loudness balance will continue to level up even after the tinnitus is masked by the testing frequency.

It is impossible for a patient with hysterical deafness or a malingerer to reproduce with any approach to accuracy the loudness balance tests. The binaural or monaural balance tests do not ordinarily require masking to eliminate contralateral audition, but the use of masking in conjunction with the elicitation of recruitment phenomena gives promise of interesting revelations.

The recruitment phenomena are not limited in application to the neural mechanism of hearing. With appropriate stimuli they may be used to study the relative intensity of sensations from other sense organs as compared to the normal. They, therefore, may be used to investigate the sensations of sight, touch, pain, and the static sense, etc.

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140 East 54th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

I.—THE CLINICAL PICTURE OF BACTERIAL MENINGITIS WITH PARTICULAR REFERENCE TO ITS CHANGING PHASES.*

DR. SAMUEL J. KOPETZKY, New York.

The average clinical picture of meningitis has been drawn from a study of the pathology as found at autopsies, which is the end picture of a lesion in the meninges or in the brain tissue itself. The reason for the title, "The Changing Phases," is because there is no fixed picture in living pathology. In dead room pathology we have a static picture.

Anyone, any nurse with ordinary experience, can diagnose the disease when the clinical picture contains all the factors of an inflammation of the brain tissue—cerebritis—and an invasion or infection with inflammatory reaction of the subarachnoid spaces, with an invasion of the circulating fluid. No one needs much knowledge to make a diagnosis of meningitis in the presence of opisthotonos, eye-ground picture, clouded sensorium, temperature, and cloudy fluid. For a long time the stress has been laid on early recognition of intracranial infection, and we have come to recognize a phase in the invasion of the cranial cavity by bacteria wherein the patient is asymptomatic; and it is in the effort to find and recognize the onset of the lesion at that time that the laboratory set-up, the details of which my colleagues on this program will elucidate, has been worked out.

Certain fundamental things, however, I may stress without impinging on their papers; namely, that an invasion of the endocranium with bacteria does not necessarily mean an inflammatory reaction of the tissues within the skull. An invasion by bacteria of the cerebrospinal system does not necessarily mean an inflammatory reaction in the tissues

*Read before the New York Academy of Medicine, Section on Otolaryngology, April 21, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 24, 1937.

comprising the dura, the pia, or the surface of the brain, nor a reaction in the blood vessel channels of these tissues. If inflammatory reactions result, one would find microscopical evidence of inflammation as a result of bacterial invasion, as one finds it anywhere else in the body. I think it is necessary that the patient's condition be recognized when *the invasion* takes place, and therapy started before infection is begun.

This change can be estimated in a detailed study of the cerebrospinal fluid, and that study must embrace the four salient points; namely, its physical qualities, the pressure under which it is withdrawn, its cytology, its bacteriology, and its chemistry or biochemistry. The findings must be studied and observations made on the circulating blood of the patient at the same time for comparison and evaluation. Then it is possible to estimate an invasion of the cerebrospinal system before an inflammatory reaction takes place.

A factor which I think has not received the significance that it warrants is the presence of headache and a mild degree of muscular pain in the muscles of the back of the neck. Transient photophobias are also significant. If you withdraw and test the cerebrospinal fluid in these cases with generalized frontal headache and do the chemistry and other examinations of the fluid and trace them in relation to the chemistry of the circulating blood, valuable information is given; and you will know when an invasion presents itself, which gives you time to do something before lesions are established from which there is no recovery. Therefore, no one would expect at this stage and at this time to have a review of a textbook picture of the changing phases of the neurological picture. It is suggested that we accept the new standpoint and separate the asymptomatic invasion of the intracranial cavity from the symptomatic.

Of course, bacteria are fed into the cerebrospinal system from a focal point from which they arise. And in all meningitis the focal point must be found and eradicated, for sterilize the fluid as you will, if it is repeatedly invaded—not necessarily infected—your therapy eventually is defeated. So it means the evisceration of the focus in the temporal bone, and when I say the temporal bone I mean the entire bone. This entails evisceration of the mastoid process, the

opening into the middle ear, and the evisceration of cellular tissue if such is present in the petrosal pyramid. The procedure includes removal of the tegmen mastoideum, tympani, and the visceral walls of the petrosal pyramid, so as to cut the venous blood channels leading from these points into the dura. It should not be forgotten that the dural venous channels are part of the skeletal system. Block these channels, and the regression thrombosis along them toward the dura, which often carries the infection to the meninges, is stopped.

In the recognition of a bacterial invasion of the meningeal system, one factor stands out; namely, the loss or drop in pH, the rise in lactic acid, the drop in chlorides and carbonates, and then the other things with which we are all familiar. Blood transfusions in small quantities, repeated every second day, restore the reserve alkalies of the tissues, to use a term which is not scientifically exact but conveys the idea. The reason all of us do not become infected by the bacteria we breathe is because we do not furnish a favorable medium for their growth. We lessen, or inhibit, the growth of these invading organisms by rendering the tissues they are invading unfavorable media for their growth. Hence the beneficial effects of small transfusions repeated every second day.

There is one thing that must be watched in the transfusions. If you are successful—and this year's reports around the city show that there have been a good many successful cases, the danger must be recognized of a too-high concentration of a patient's blood, the hemoglobin rising to a danger point so that a generalized thrombosis may occur. It is necessary, therefore, when giving these repeated transfusions, to make careful serial estimations of the hemoglobin. When it gets too high it is time to stop; but if the fluid is not yet sterile and the hemoglobin is high, one can transfuse with blood serum alone.

With this, purely as an introduction, I leave the subject for those who are going to present it to you in detail.

71 East 80th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

II.—THE PATHWAYS OF INFECTION FROM THE PARANASAL SINUSES.*

DR. RUDOLPH KRAMER, New York.

The study of the pathways of infection from the paranasal sinuses to the meninges is not only of academic interest, but it has practical value in the prophylaxis and therapy of bacterial meningitis. I need only cite the results obtained in the treatment of frontal bone osteomyelitis originating from frontal sinusitis. These results are due to greater knowledge of the pathways of infection from the frontal sinuses to the cranial contents.

There are some pathways of infection which are either common knowledge of rhinologists or are depicted in the literature. The first pathway of this type is by way of osteitis; for example, that involving the posterior wall of the frontal sinus. This is familiar to rhinologists as a finding at operation. The second pathway is through a fracture involving the parasanal sinuses. A third method of spread is by way of osteomyelitis, particularly of the frontal bone. This has been very thoroughly described by Furstenberg and by Mosher. I can most earnestly recommend their articles on this phase of the subject for study.

My presentation is concerned with the demonstration of other pathways of infection which, while they are mentioned in the literature as theoretical possibilities in some instances and in other instances have been demonstrated in isolated cases, have been hitherto either unproved or the evidence by microscopy has been generally unfamiliar.

The lantern slides illustrating the various pathways of infection are from some of the cases at Mount Sinai Hospital which were collected and studied by one of the members of my staff there, Dr. Max Som, and myself.

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*Read before the New York Academy of Medicine, Section on Otolaryngology, April 21, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 24, 1937.

We have so far considered the following definitely determined paths:

1. Osteitis.

2. Osteomyelitis.

3. Phlebitis and the possibility of admixture of these three processes.

4. Direct spread through dural and neural polyp.

5. Defect in the cranial walls of the sinuses.

6. Perivascular lymph space spread.

7. Perineural lymph space spread.

In addition, we have clinical evidence but not proof by microscopy of the following additional pathways:

1. Orbital infection from sinusitis with a subsequent spread either by vascular involvement or by direct purulent or cellulitic spread through the orbital fissures into the cranial cavity.

2. Involvement of the pterygomaxillary fossa from sinusitis and either direct extension through the basal foramina or by vascular involvement, such as pterygoid plexus phlebitis.

3. Extension from the maxillary sinus through osteomyelitis of the superior maxilla, then either through the orbit or through the frontal bone, or by way of the blood vessels.

4. Metastases from a distant focus, such as osteomyelitis of an extremity due to fracture, to the walls of a paranasal sinus and subsequent osteomyelitic extension to the meninges.

121 East 60th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

III.—DIFFERENTIAL DIAGNOSIS OF SUPPURATIVE MENINGITIS CAUSED BY PARANASAL SINUS DISEASE. WITH SOME SUGGESTED PROPHYLACTIC MEASURES.*

DR. CHARLES J. IMPERATORI, New York.

It is accepted that there are three pathways of infection of the meninges, *viz.:* by way of the venous blood stream, direct extension through the bone, and by the olfactory perineural sheaths. They are proportioned in occurrence as approximately 48%, 43%, and 9%, respectively.[1]

However, in the further analysis of the venous blood stream infection resulting in cavernous sinus thrombosis, eight cases of the 22 cited were by direct extension from the frontal, ethmoid or sphenoidal sinuses through their osseous veins, so that for practical purposes of diagnosis, we may consider that the predominating source of infection is through the paranasal sinuses, and we must therefore take them into careful account in our attempt to diagnose the cause of the infection.

Other sources of primary foci inducing a suppurative meningitis through the blood stream may occur on the face, pharynx or middle ear. Erysipelas, cellulitis of the face, pustules within the nose, and orbital abscess must be considered and eliminated as a possible cause of the symptoms. Following a nasal operation, or a severe head cold with profuse purulent nasal discharge, symptoms, physical signs and laboratory findings such as those enumerated below are indicative of an invasion of the meninges:

Headache; as a rule, persistent and boring in character.

High temperature, pulse and respiration; at times the pulse and respiration are slowed or irregular.

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Vomiting: The vomiting occurring in acidosis is differentiated by the physical and neurological findings; vomiting in scarlet fever is projectile and there is usually an angina and the strawberry tongue.

Photophobia, and in many instances, sensitiveness to extraneous noises.

Irritability and restlessness.

Neurological signs.

Cervical stiffness. Kernig. Brudzinski.

Spinal fluid studies, both physical, chemical and bacteriological, are of utmost importance. By these studies we may be able to differentiate a meningococcus intracellularis meningitis or tuberculous meningitis. Pnueumonia with a meningism will be ruled out by the spinal tap and confirmed by physical signs and X-ray.

The chemical analysis of the spinal fluid and the estimation of the amount of sugar is important, for by this means we are greatly helped. Particularly if there is a high cell count and no demonstrable bacteria, we are justified in considering the condition as a localized meningitis and that proper surgical intervention at the focus of infection may result in a cure of the patient.

Blood counts and blood cultures will help indicate and may differentiate, the infection and its source.

X-ray examination in the early stages of meningitis, in my experience, has been confusing. Occasionally edema of the Pacchionian bodies and surface markings of the meninges are misinterpreted.

An otological examination must be done; otitis media, mastoiditis, labyrinthitis and petrositis must be differentiated.

Injuries of the head may not have any external evidence, and degrees of concussion may be difficult to differentiate from a bacterial invasion of the meninges, at least until further pathological changes have occurred.

In acute poliomyelitis in the preparalytic stage, headache, vomiting and photophobia may obscure the diagnosis for a time. Spinal tap and neurological findings help differentiate.

Syphilis and malignant disease must be differentiated.

The number of spontaneous cases following paranasal sinus disease is small in comparison with aural disease, the proportion being about 8-200.

In an analysis of 17 cases of rhinogenic meningitis that occurred at the New York Post-Graduate Hospital in the past 10 years, seven complicated paranasal sinus disease and were operated upon by the external route, seven followed intranasal operations on the sinuses, two followed submucous resections, one followed cavernous sinus thrombosis due to a nasal furuncle, and one occurred following an acute fulminating ethmoiditis without any operative interference other than to remove necrotic bone from the frontal and malar region after the subsidence of the meningitis. Nine cases were infected by the staphylococcus, five by the streptococcus, and two by the pneumococcus. One case recovered and no organism was demonstrated in the spinal fluid.

SUGGESTED PROPHYLAXIS AGAINST SUPPURATIVE MENINGITIS.

The factors that have to be considered are, to a degree, dependent on so many contingencies that it becomes necessary to divide them into three groups:

1. Unavoidable Causes: Such as congenital defects of the bony case of the brain, nasal glioma, fulminating sinusitis during influenzal epidemic, accidental injuries, and other complicating infections of the brain or meninges following pneumonia, bronchiectasis, etc., by bacteria-laden emboli.

2. Partially Avoidable Causes: Surgical trauma.

a. Loss of orientation during operation on the sinuses, resulting in perforation of the brain case, particularly fracture of the cribriform and direct injury to the meninges.

b. Trauma associated with bungling operative methods on the nasal septum or middle turbinate, and especially cauterization of the latter.

c. Incomplete operation on the sinuses, resulting in failure of drainage. Prolonged nasal packing and repeated and unsuccessful attempts to remove a foreign body from the nose.

3. Avoidable Causes:

a. Certain types of operation on the ethmoid sinuses, and particularly the Sluder and the Ballenger intranasal ablation

en masse of the ethmoid masses. Excepting in the hands of these authors, this method of operating has been the direct cause of meningitis in many instances. This method should be discarded.

b. Intranasal operations on the frontal sinus. The external operation should be the method of choice, after conservative methods have failed. By this is meant mucosal shrinkage and attempted re-establishment of drainage.

c. Prolonged operating—so-called "through operating"—on patients with acute fulminating frontal or ethmoidal involvement. This is especially to be avoided in children or young adults in whom the infection is due to swimming and diving. Operations on these types of infections should consist of an external incision, with an opening into the sinuses for drainage.

In these types of infection, comparatively little pus is found, but there is intense turgescence of the blood vessels. Excessive disturbance of this infected field results in emboli into the general circulation. These infections are staphylococcic in the majority of instances.

It is unnecessary to add that uncertainty of landmarks of safety and loss of way in the intricacies of the normal anatomy of the location and relationship to vital structures make any intra- or extranasal procedure exceedingly hazardous.

Any other methods that may be considered as prophylactic are, in my opinion, of doubtful value, for the infection has already occurred.

The use of sprays or applications has been proven to be of value in experimental studies done on animals in whom the virus of poliomyelitis was instillated into the upper airways. The percentage of prevention is so striking that it must be accepted. and digression from the subject under immediate discussion will take but a few words.

Sabin and Olitsky[2] have definitely shown in their experimental studies that the virus of poliomyelitis has its portal of entry by way of the olfactory bulbs. Prophylactic sprays such as advised by the U. S. Public Health Service in pre-

venting poliomyelitis should receive consideration. The recommendation is to use a spray consisting of ½% alum and ½% picric acid solution. Others have advised the use of 1% zinc chloride solution.

To return to the subject under discussion:

In some individuals there is undoubtedly a structural virus barrier in the lining membranes. This may be inherent in the individual, or due to some previous involvement of these tissues, or by chemical means, that result in infiltration of the tissues. In any event, it has been shown by Fenton and Larsell[3] that various antiseptics produce a reaction of tissue infiltration with round cells and histocytes, and without a tendency to repair the musocal surfaces.

SUMMARY AS TO PROPHYLAXIS.

Septal operations should not be done in the presence of acute infective conditions. In all septal operations especial care should be exercised in the partial removal of the perpendicular plate of the ethmoid. Using a biting forceps in its removal, and twisting and rocking movements, should be avoided.

Polyps should not be removed during an acute infection.

In those acute fulminating cases of sinus disease, the external operation is to be preferred.

At all times careful intranasal operating.

It is better to stop operating if the landmarks are lost or obscured.

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108 East 38th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

IV.—DIFFERENTIAL DIAGNOSIS OF EXTRAOTITIC MENINGITIS.*

DR. E. D. FRIEDMAN, New York.

Extraotitic meningitis may occur in any severe infection and in many forms of intoxication (meningismus). It may be purulent or nonpurulent. Among the nonpurulent forms, tuberculous meningitis is the most important. It usually occurs in children. For weeks before the acute syndrome there is a change in the mood and attitude of the patients. They cease to play, complain of pain in the legs, sit quietly by themselves, and occasionally complain of headache; there is poor appetite and nausea with occasional vomiting and drowsiness. Diagnosis of gastric disturbance is frequently made. After a short time there is the sudden outburst of meningeal signs and symptoms, with the hydrocephalic cry which wakes the child from sleep. Children with vague digestive disturbances and alterations of mood should not be considered cases of acidosis, but rather should arouse the suspicion of tuberculous meningitis. There is usually an irregular fever, and the march of events continues rapidly, with death in about three weeks.

The pathological lesion is most pronounced at the base, hence the frequency of ocular palsies. The exudate follows fissures and pial vessels. It may be part of a general miliary process (indicating the importance of an X-ray examination of the chest), or it may occur independently following an old tuberculoma of the brain. There may be tuberculous foci in the glands, in the bones, or lungs. There may be tuberculides in the skin. Cases of tuberculous meningitis have been described in children who have been exposed to a tuberculous servant in the home.

Frequently, in the early stages, we find a slow, irregular pulse, and leucopenia with a tendency to polynucleosis. There

*Read before the New York Academy of Medicine, Section on Otolaryngology, April 21, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 24, 1937.

may occasionally be a purulent exudate in tuberculous meningitis (in the rapidly fatal cases), but in most instances the spinal fluid is clear, under increased pressure, and the protein is increased; there is a pleocytosis consisting chiefly of lymphocytes. A coagulum occurs in the form of a fine filament in the clear spinal fluid of tuberculous meningitis, and in this filament the tubercle bacillus can be demonstrated. In tuberculous meningitis the sugar content falls and the chloride content is below 600. There may be a positive tryptophan reaction.

The deep reflexes are diminished, especially the knee-jerks. There is frequently retention of urine. In very young infants there is a tense fontanelle. Tubercles may occasionally be found in the choroid.

The most common pyogenic form of extraotitic meningitis is the epidemic form. This usually appears acutely, with chills, fever and vomiting. Spinal fluid is characteristically cloudy and contains gram-negative organisms. The blood culture may be positive in the septic cases. These may occur without meningeal signs. In some cases the fluid does not show the bacteria early in the disease, but it must be remembered that the culture may be spoiled if the fluid is cooled off in transportation.

The clinical picture is differentiated from tuberculous meningitis by more constitutional disturbance and more pronounced meningeal signs. We find, in the most severe cases, excessive perspiration, swelling of the joints, herpes, and purpuric spots; hence, the name spotted fever. One can differentiate this disease from typhus by demonstrating the organism in meningitis and by the Felix Weil reaction in typhus.

Pathologically, epidemic meningitis resembles the pneumococcic form. In both instances the convexity of the brain is chiefly involved.

Focal signs in epidemic meningitis are usually not basilar in type, although I have occasionally observed cases of this type of meningitis with oculomotor palsies suggestive of a basilar process.

Choked disc may occur from hydrocephalus. Difficulties in hearing, deafness and blindness may occur as sequelae of the disease. There may also be dementia.

If the origin of infection cannot be determined in a case of purulent meningitis, it is usually caused by the meningococcus; hence, give serum immediately and continue to do so until the organism is identified. In meningococcus meningitis there is first an upper respiratory infection. This leads to a blood stream infection and finally to meningitis. The organism is gram-negative. It secretes chiefly an endotoxin. There are several types of organisms, which accounts for the varying effects of serum. In the septic cases there is a typical rash plus arthritis.

I recall the case of a child who was admitted to the hospital in stupor. She presented signs of a fulminating sepsis, with diffuse petechial rash, a positive blood culture with organisms crowding the leukocytes in the blood smear.

If the meningeal picture is prolonged beyond seven days, treatment is inefficient. The prolongation of symptoms may be due to loculation of fluid. There are occasional atypical cases with prolonged fever, arthritis, endocarditis, otitis and subarachnoid block. In the early stages the spinal fluid may be normal. Later, as the process heals, the fluid becomes clearer, there is less albumin, more sugar, etc.

The diagnosis is easy in epidemic times. This form of meningitis must be differentiated from:

1. Meningismus, which accompanies acute infections (fewer cells). Serous meningitis is accompanied by a clear meningeal effusion without organisms. This may be due to hypersecretion or defective absorption (otitic hydrocephalus). Serous meningitis (the term suggested by Quincke) shows fluid under pressure and increased albumin content. The distinction between meningismus and serous meningitis is still not clear. Quincke's serous meningitis has sometimes been called a nontuberculous hydrocephalus. It may be due to:

a. Trauma; *b.* toxic factors (alcoholic wet brain); *c.* angioneurotic edema; *d.* venous stasis.

2. Sepsis. (I recall a case of viridans sepsis with evidence of multiple embolic lesions in the brain which were accompanied by a low grade purulent meningitis.)

3. Cerebral poliomyelitis.

4. Tuberculous meningitis.

5. Encephalitis.

6. Subarachnoid hemorrhage.

7. Lues with meningeal signs. Luetic meningitis is chiefly basilar; bilateral facial palsy; diabetes insipidus.

8. Sympathetic meningitis.

9. Unusual forms, such as those due to influenza, torula, or actinomycosis.

10. Trauma to skull (microscopic fracture).

11. Occasionally an aseptic purulent meningitis may follow the introduction of air or serum, and spinal anesthesia.

12. Suppurative epimeningitis, due to staphylococcus: No cerebral symptoms. Meningeal signs limited to the lower part of the body. Sensitive pressure point over the spinal column. Demonstrations of staphylococci in purulent spinal fluid.

13. Subdural hematoma.

1192 Park Avenue.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Philadelphia, June 7 and 8, prior to the meeting of the American Medical Association. Ninety-four candidates were examined; of this number, 72 were certified.

An examination will be held in Chicago, Oct. 8 and 9, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificate should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha.

SYMPOSIUM ON BACTERIAL MENINGITIS.

V.—THE NEW YORK MENINGITIS COMMITTEE OF THE AMERICAN OTOLOGICAL SOCIETY.*

DR. THOMAS J. HARRIS, New York.

The American Otological Society has for many years been giving much attention to the subject of progressive deafness, especially otosclerosis, through its Research Committee. Important as this is, the Council of the society felt that even more important was a systematic study of everything that could be learned in connection with otitic meningitis, a condition which has caused great loss of life and which has baffled all investigations up to the present time.

At the annual meeting of the society three years ago, held at Atlantic City, the president, Dr. George Tobey, of Boston, proposed that the society undertake a systematic investigation of purulent meningitis of otitic origin and that special committees should be organized in the various metropolitan centers in the country which should head up such an investigation. Such committees have been organized in this city and in Chicago—in this city under the leadership of Dr. James G. Dwyer. A questionnaire covering all phases of the subject has been prepared and widely distributed. It is the wish and intent, dependent upon the invitation of the doctor in charge of the case, that a member of the committee should see every case of meningitis in Greater New York. A considerable number of cases have been collected in the last two years and reported at the national meetings of the society; but a much more widespread campaign is necessary.

The committee, therefore, earnestly seeks the co-operation of the members of this Section. Numbering as it does something over a 100 members—probably nearer 200—it could give great assistance in this investigation by promptly reporting to the chairman, Dr. James G. Dwyer, any case

*Read before the New York Academy of Medicine, Section on Otolaryngology, April 21, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 24, 1937.

of meningitis that may occur and inviting one of the members of the committee to see the case. The Council of the Society felt that each local committee should have associated with it neurologists and neuropathologists.

I think that this meeting tonight is one of the most important in a long time. To me it represents an aroused sentiment on the subject of meningitis. Such papers as we have listened to will carry tremendous weight throughout the country.

Some of the questions that have come before the committee have been debatable. We are frank to admit that our views are not held by everyone; but the more knowledge we can get, the better can we arrive at some solution. This Section can very properly interest itself in this campaign. Perhaps we could have a meeting every year on the subject of meningitis, and a committee of the Section could be organized to study the problem and report its findings. In this way we would be assisting in carrying on the investigation of this clinical problem, and after such meetings as this one tonight it would seem to me that a solution must be attainable.

104 East 40th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

VI.—A SUMMARY OF METHODS USED IN TREATING MENINGITIS SECONDARY TO INFECTIONS OF THE EARS AND SINUSES.*†‡

DR. JOSEPHINE B. NEAL, DR. HENRY W. JACKSON and DR. EMANUEL APPELBAUM, New York.

Meningitis secondary to infections of the ears and sinuses may be caused by the streptococcus, pneumococcus, influenza bacillus, occasionally the staphylococcus or by a combination of two or more of these organisms. Rather infrequently other organisms are involved, as the streptothrix, B.Coli, B.Friedlander and the torula. When meningitis follows pneumonia or upper respiratory infections it is quite probable that the meningitis often is secondary to a sinusitis. In certain instances it may be that there is a primary bacteremia with subsequent or perhaps simultaneous localization in the sinuses, middle ear or mastoids and meninges.

The longer we observe these cases of meningitis presumably secondary to a primary focus of infection, the more we become aware of how inadequate is our knowledge of the relationship. We believe that some light could be thrown on this subject if more careful bacteriological studies were made of these foci of infection during life and particularly at necropsy.

Many attempts have been made to treat these forms of meningitis. They may be roughly grouped under three heads: *1.* Surgical procedures. *2.* Serological and allied therapy. *3.* Chemical agents.

1. Surgical Procedures: We are using the term surgical procedures in a broad sense. The removal of foci of infection at the earliest possible moment is clearly indicated. It is of course evident that it is often difficult, if not impossible,

*Read before the New York Academy of Medicine, Section on Otolaryngology, April 21, 1937.

†Division Applied Therapy, Bureau of Laboratories, Department of Health, New York City.

‡We wish to thank the Winthrop Chemical Company for generously supplying us with Prontosil, Prontylin and Prontylin crystals.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 24, 1937.

to completely eradicate these foci. In this case some degree of drainage by other methods should be attempted.

Various forms of drainage have been tried. In our experience the more radical methods of establishing drainage, such as laminectomy or trephining the cisterna magna, have no advantage over repeated lumbar punctures or cisternal or ventricular punctures if block occurs. Indeed, establishing permanent drainage may be a disadvantage if serum or chemicals are to be used intraspinally. Forced spinal or perivascular drainage has been used rather extensively during the past four or five years. We have had considerable experience with this method of treatment. As a result of our experience we do not recommend it. We believe that little if any more spinal fluid can be obtained in this way than by leaving the lumbar puncture needle in place for the same length of time and adequately supplying the patient with fluid by mouth. Furthermore, there is in our opinion, no advantage in continuing the drainage for several hours. We do know that in spite of this method being used the spinal fluid not infrequently becomes so purulent that drainage is impossible. In certain instances the necropsy showed an extensive edema at the base of the brain, although there appeared to be free drainage of fluid before this method was applied. It has been reported to us that in certain instances of tuberculous meningitis treated by forced drainage the brains showed hydrosis at necropsy. These statements of course apply only to the use of forced spinal drainage in meningitis.

2. Serological and Allied Therapy: Serums in the treatment of meningitis other than the meningococcic have given disappointing results. In pneumococcic meningitis we have used specific serums frequently without a single recovery; however, in a very few instances there have been reports of recoveries following administration of serums.

In streptococcic meningitis we have used antistreptococcic serum with occasional recoveries. Usually we did not believe that the recovery was due to the serum. In two cases, however, in which a hemolytic streptococcic meningitis followed scarlet fever complicated by otitis media, we believe we were justified in attributing the recoveries to the specific action of the antiscarlatinal serum. When a streptococcic meningitis follows scarlet fever we think it is advisable to use either the antiscarlatinal or convalescent serum.

The results with the use of the specific serum in influenzal meningitis, both in cases that seemed to be primary and those that were presumably secondary, have been on the whole unsatisfactory. As a rule there has been a period of improvement, and in a few instances recovery has taken place; therefore, we continue to use this serum. It is possible that in the future a more potent serum may be developed.

In cases of meningitis due to the staphylococcus, organisms of the colon group, the pyocyaneus, etc., bacteriophage should be used if available.

We have also used autogenous vaccines, both intraspinally and subcutaneously, in a small number of cases. Two recoveries, one a staphylococcic meningitis and one an influenzal meningitis—followed the use of this method. In most instances, the course of the meningitis is too short to admit of this treatment.

3. Chemical Agents: In the past the use of various chemical agents has been advocated by a number of workers. We have injected intraspinally gentian violet, acriviolet, acriflavin and ethylhydrocuprein hydrochloride (optochin), both alone and in combination with serums. It is our impression that these agents are of doubtful value and occasionally may be harmful. We have, therefore, discontinued their use with the possible exception of optochin in pneumococcic meningitis. There has been a tendency of late to employ the intracarotid route for the injection of serum and chemical agents. We have never used nor advocated this method. In the first place, we can see no advantage from its use, since the rapidity of the circulation of the blood is so great. In the second place, one case has been under our observation and others have been reported to us in which thrombosis of the cerebral vessels followed this method of injection. We therefore regard it as a dangerous procedure.

In recent months the use of para-aminobenzenesulphonamide has received widespread attention. It is not within the scope of this paper to discuss in detail the history and the experimental background of this chemical. It has been shown by work with animals that it is highly effective in its action on the hemolytic streptococcus,[1] the meningococcus[2] and the pneumococcus. Its effect on other organisms appears doubtful, but work has just been begun which seems to show that it

may act in infections with the influenza bacillus. Clinical studies at present do not seem to confirm the results of animal experimentations with the pneumococcus. The mode of action of this chemical is not understood. For instance, in vitro it is reported by several workers to be more highly bacteriostatic and bactericidal for the pneumococcus than for the streptococcus. In man, however, the reverse is true. In passing, it may be noted that certain strains of the hemolytic streptococcus hemolyze only on horse blood plates, and on media which do not contain glucose. The whole question is being actively investigated and new information is rapidly accumulating.

We have used Prontosil and sulfanilamide usually in the form of Prontylin in cases of meningitis due to the hemolytic streptococcus, the pneumococcus and the influenza bacillus. We have also used this chemical in a few cases of meningococcic meningitis and meningococcemia, but this does not pertain to the present discussion.

We have treated nine cases of pneumococcic meningitis. At present only one has recovered. This was a case of type 31 which developed within 24 hours following an ethmoid operation and tonsillectomy. Seven have died and one is still under treatment.

Two cases of influenzal meningitis have received this chemical agent in addition to the specific serum with one recovery.

The use of Prontosil and Prontylin in cases of hemolytic streptococcus meningitis have yielded results that seem to us quite astounding. While the number of cases is still small, the results have been almost uniformly favorable. We have had twelve cases. In every instance the diagnosis was made by culturing the hemolytic streptococcus from the spinal fluid. All but one of these have been secondary to infections of the ears or mastoids; this patient had a pansinusitis. Nine have recovered, two have died and the twelfth is still under observation. One fatal case had a double otitis media and a double mastoiditis. There was also clinical evidence of a brain abscess and the dural plate was found to be nearly destroyed when the mastoid operation was performed. The patient died within 12 hours after the first dose of Prontosil. The second case was one of severe pansinusitis of long standing

and the meningitis was of several days duration. The patient was in a grave condition and died within 24 hours after the Prontosil was given.

During the period of more than 26 years that the meningitis division has been in existence, the case fatality in hemolytic streptococcic meningitis has been uniformly very high. Up to the end of 1936 we had seen 274 cases of all kinds of streptococcic meningitis, most of which have been of the hemolytic variety. Of these cases, only 15 recovered of which nine were definitely caused by the hemolytic streptococcus. There was a tenth recovery in which there was a mixed infection of the meningococcus and the hemolytic streptococcus. Three of these recoveries were in patients in whom the meningitis developed following scarlet fever. In two instances the antiscarlatinal serum was used and in the third large amounts of convalescent serum and small amounts of Prontosil and Prontylin.

One may wonder if we are dealing at present with less virulent strains of the hemolytic streptococcus. Experiments have been started to determine if this is so. It would seem remarkable if there has been a sudden change in virulence. During the year 1936 we saw 20 cases of hemolytic streptococcic meningitis, all of which died with the exception of one following scarlet fever, to which we have already referred. The dosage of sulfanilamide is still more or less empirical. We have advised the use of rather moderate amounts, 5-10 cc. of Prontosil every four hours and 5-15 gr. of Prontylin every six hours. In children we have generally followed the recommendations of Long and Bliss of giving 1 cc. of Prontosil per pound of body weight in 24 hours. In two of the fatal cases of pneumococcic meningitis an .8 per cent solution in normal saline of Prontylin crystals was injected into the subarachnoid space. Since it has been shown by Marshall, Emerson and Cutting[4] that after oral administration the concentration of sulfanilamide in the spinal fluid is nearly equal to that in the blood, the intraspinal injection would seem to be unnecessary.

There has been considerable discussion of the toxic effects of sulfanilamide. We have not observed such effects in any alarming degree. We realize of course that an occasional patient may be as unduly sensitive to this as to any other

chemical. It is advisable to make repeated blood counts during the period of treatment.

In conclusion, we wish to emphasize the following points in the treatment of these forms of meningitis: *1.* Foci of infection should be removed as early and as completely as possible. *2.* Early and repeated drainage of the spinal fluid is very important. *3.* The use of specific serums may at times be of value. *4.* Bacteriophage is recommended when available. *5.* Sulfanilamide is a most valuable contribution to the treatment of meningitis due to the hemolytic streptococcus. It may be of value in other forms of purulent meningitis secondary to infections of the ear or sinuses.

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60 Gramercy Park Avenue.

210 East 68th Street.

50 West 96th Street.

SYMPOSIUM ON BACTERIAL MENINGITIS.

VII.—THE PATHWAYS OF INFECTION FROM THE EAR.*

DR. MARVIN F. JONES, New York.

Dr. Kopetzky has emphasized a very important part of the presentation that I was about to make. In other words, he stole the thunder. Since he has detailed the anatomic pathways of infection, it would be an old story to all of you if I were to continue that topic. When I talked to Dr. Fowler about my 10-minute discourse, I suggested to him that it would be better to outline a course which would deal surgically with the problem of bacterial meningitis, rather than to enumerate the anatomic pathways.

Those of us who have done mastoidectomies and been unfortunate enough to have our patients die have examined these temporal bones at autopsy, and the strange thing is that some do get well. We are brought face to face with the fact that cells exist in the mastoid after a supposedly complete exenteration has been done, cells which harbor an infection which has been advanced through the bone by way of the blood vessels or sometimes by way of the nerves. There is a fulminating type of meningitis that seems to progress no matter what we do. And, of course, there is the typical bacillary meningitis which we find so commonly, particularly in children, where there has been otitis media and an early involvement of the mastoid has so devitalized the structures that no resistance is offered. The erosion produced by a very low-grade infectious process converts all the mastoid process into one large cavity. A superimposed acute infection rapidly progresses to an acute meningitis and death of the patient.

When a unilateral headache is persistent, or when a child wakes up at night with a headache, the headache should

*Read as part of the Symposium on Bacterial Meningitis, New York Academy of Medicine, Section on Otolaryngology, New York, April 21, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 6, 1937.

always be a danger sign. In such cases we will have to put our pride in our pockets and recognize that we have left infected cells, and that a headache and profuse continuing discharge mean that we have certain work to do, and that is to go back into the mastoid and find out where the trouble is.

There is a well recognized dividing line between meningeal symptoms with increased pressure in the spinal fluid and increased cell count but no organisms present in the fluid, and the cases where the organism is demonstrable. These cases are not recognized as being true meningitis cases but are really a prodromal stage of meningitis. A large percentage of them will develop meningitis. In this stage much can be accomplished by surgery, and this includes following out the cell pathways and discovering the tract of the infection.

LIGATION OF INTERNAL JUGULAR VEIN IN LATERAL SINUS THROMBOSIS.*†‡

DR. JOEL N. NOVICK, Washington, D. C.

One of the dreadful complications of middle ear and mastoid disease that the otologist often confronts is lateral sinus thrombosis. This catastrophe, not infrequently coming on in the course of an acute or chronic otitis media, acute or chronic mastoiditis, or following a mastoidectomy, throws the surgeon into an uncomfortable state of mind. He knows that the patient is acutely and dangerously ill, and that something has to be done; but as to the course of action he is not clear.

His predecessors have established certain surgical procedures in the management of this complication, namely, removal of the clot and ligation of the internal jugular vein; but they have fallen short of proving the efficacy of such treatment. His contemporaries, moreover, are not at all in harmony on the question of ligation and are aligned into two schools, the pros and cons of this procedure.

Before considering the evidence in favor of, and that against ligation of the internal jugular vein in a case of lateral sinus thrombosis, it seems advisable to review the following: *1.* Anatomy of the lateral sinus and its tributaries. *2.* Physiology of the blood circulation through the brain. *3.* Etiology and mechanism of thrombosis. *4.* Types of thrombi and sequellae.

Piersol, in discussing the anatomy of the lateral sinus, uses the terms transverse sinus and lateral sinus interchangeably. Quoting: "The transverse sinus takes origin opposite the internal occipital protuberance, at which point there is a meeting of the five sinuses, the two lateral, the superior sagittal, the straight and the occipital. From this meeting point,

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*Thesis for M.Sc. (M.D.) degree at Graduate School of Medicine of University of Pennsylvania, June, 1936.

†Department of Oto-rhino-laryngology, George Washington University, School of Medicine.

‡Elaboration on paper read before Philadelphia Laryngological Society, May, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 8, 1937.

which is termed 'confluens sinum,' each transverse sinus passes laterally over the squamous portion of the occipital bone along the line of attachment of the tentorium cerebelli, and passing over the posterior inferior angle of the parietal bone is confined medially upon the inner surface of the mastoid portion of the temporal bone and the jugular process of the occipital to reach the jugular foramen, where it opens into the internal jugular vein. As it passes upon the mastoid portion of the temporal bone, it leaves the line of attachment of the tentorium cerebelli, passing somewhat downward as well as medialward, and follows the line of junction of the petrous and mastoid portions of the bone in a somewhat 'S' shaped course, whence this portion of it is frequently termed the sigmoid sinus.

"Throughout that portion of their course in which the transverse sinuses lie in the line of attachment of the tentorium cerebelli, they are triangular in cross section, but in their sigmoid portion they are semicircular, with flat surface against brain and convex against bone.

"The right sinus has a diameter of 9 to 12 mm., while the left varies from 3 to 5 mm.

"Considerable variation exists in the relative sizes of the left and right transverse sinuses, in accordance as the superior sagittal sinus opens more or less directly into one or the other. The tendency is for the superior sagittal sinus to open into the right transverse sinus, though quite often it opens into the left, and occasionally communicating equally with both. In 100 crania Rudiages found that the right transverse sinus was the larger in 70 cases, the left in 27, and both equal in size in only three cases.

"The knee of the sigmoid portion of the transverse sinus extends further inward on the right side than on the left, and this fact, together with the larger size of the right transverse sinus as compared to the left aids in explaining the greater frequency of thrombosis of the right transverse sinus.

"The transverse sinus communicates with the cavernous sinus by way of the two petrosal sinuses: the inferior petrosal, the larger of the two, opening into the transverse sinus at the blub of the jugular vein; the superior petrosal, the smaller of the two, opening into the transverse sinus just

as it leaves attachment in the tentorium cerebelli to become the sigmoid sinus. Other tributaries are:

"*1.* Mastoid emissary vein which connects the sigmoid sinus with the posterior auricular or occipital vein.

"*2.* Posterior condyloid emissary connecting the vertebral vein with the sigmoid sinus.

"*3.* Internal auditory veins.

"*4.* Posterior inferior cerebral and inferior cerebellar veins."

Of interest to our discussion is the physiology of the venous circulation through the base of the skull; but since, as in other organs, the venous circulation is dependent upon that of the arterial system, we must include the latter in our discussion. Macleod and others are of the opinion that the brain, encased as it is in a rigid cranium, is unable to contract and expand in response to vasoconstriction and vasodilatation. Consequently, they argue, that the volume of blood through the brain, at any given time, cannot be materially altered; yet, we know that the blood supply to this organ does vary considerably from time to time, in health and in disease. This is made possible through a special arrangement in relationship between the venous and the arterial sides of the cerebral circulation. When there is an increase in the volume of blood on the arterial side, at any given time, there is also a corresponding decrease in the volume of blood on the venous side, the latter being brought about by an increased velocity of outflow of blood through the venous side. Thus, during an effective cardiac systole the arteries of the brain dilate to accommodate the large amount of blood. The blood from the veins is squeezed out and forced into the venous sinuses. This brings about a rise in pressure on the venous side resulting in an increased velocity of outflow of the blood from these channels.

The reverse condition, by similar reasoning, also holds true. Thus, in decreased cardiac output there is a diminished amount of blood pumped into the arterial side of the brain, a decrease in pressure on the venous side, resulting in a slowing up of the velocity of outflow through the venous sinuses.

The cerebrospinal fluid is thought to exert very little influences on the blood circulation through the brain, since the former is but small in amount, 60 to 80 cc. in man.

With regard to sinus thrombosis it is generally agreed that the following conditions are essential for its production: *1.* Slowing up of the circulation. *2.* Changes in the endothelial lining of the vessel, with roughening of the intima. *3.* Alteration in the composition of the blood.

The blood stream at this part of the venous highway is normally retarded by virtue of the widening of the stream, its tortuosity, and the emptying of its tributaries in a direction opposite to that of the stream. In disease, with less effective cardiac output there is, as explained above, a decrease in pressure on the venous side, resulting in a retardation of the outflow through the sinus. Then again, it has been definitely established that an operation slows up the depletion of the general condition of the patient, lowering of the blood pressure, the loss of blood, and weakening of the cardiac impulse.

Changes in the endothelial lining of the vessel may be brought about either by accidental injury to the sinus during operation or by infection extending into the sinus from the infected mastoid, the latter usually producing a phlebitis of the vessel.

As to the alteration in the composition of the blood there seem to be various opinions. Among changes mentioned are dehydration, increased blood viscosity, changes in acid base reaction, increased fragility of red cells, accelerated sedimentation, increased number and fragility of the platelets. The last change is probably the one more directly related to thrombosis.

In considering thrombosis we must keep in mind the differentiation between this process and that of coagulation. Coagulation is a function of stagnant blood and is brought about by virtue of ferments, which are liberated by the cellular elements, and which activate the thrombin in the plasma. The thrombin acts upon the fibrinogen and converts it into fibrin, the latter in turn enmeshing all the formed elements in the blood into a homogeneous mass. Thrombosis, on the other hand, is a function of moving blood. It does not depend upon any ferments or coagulants; in fact. even the addition of anticoagulants to the blood, as has been demonstrated by Loeb, Zurhelle, and Schwalbe, fails to check thrombosis. Thrombosis is entirely dependent upon the coaglutination of

the blood platelets along the vessel wall and the superimposition of the white blood corpuscles on the platelets. That the thrombus takes origin exclusively from the platelets has been demonstrated in the work of Zahn, Eberth, Schimelbush, and Ferge. These platelets, according to Ferge, form a system of lamellae, more or less parallel and transversely to the long axis of the vessel. These lamellae grow outward from the vessel wall by continuous deposition of platelets from the blood as the latter flows by. Eventually there results a narrowing in the lumen of the vessel, with a consequent slowing of the stream. When this occurs the leukocytes, being of lower specific gravity than the other elements of the blood, travel to the margin of the vessel and are deposited on the platelet lamellae. This further decreases the lumen and finally brings about a complete occlusion of the vessel. Then the column of blood between this white thrombus and the next anastomotic vessel undergoes coagulation and is transformed into a red clot similar in all respects to that found in postmortem changes. Frequently the distal part of the white thrombus becomes infiltrated with red blood cells and a mixed thrombus results.

From the above discussion it becomes evident that histologically there are three types of thrombi:

1. Red thrombus: Similar to a postmortem clot and a consequence of a sudden stoppage of circulation.

2. White thrombus: Consisting of coaglutinated platelets and leukocytes, and is formed slowly in actively circulating blood.

3. Mixed thrombus: Where red blood cells become incorporated in the white thrombus.

Anatomically thrombi are classified into:

1. Mural or *Incomplete:* Where the thrombus is lying against the vessel wall and does not completely obstruct it.

2. Obstructive or *Complete:* Where the thrombus completely obliterates the lumen of the vessel.

This classification seems to be hardly essential since the degree of thrombosis is influenced by the time factor, and once taking origin it will continue to increase in size till the entire lumen is filled and obstruction is complete. The

exception to this sequence is a case of an acute fulminating type of infection with a very virulent organism, where there is a rapid invasion of the blood stream, and the patient succumbs before a protective thrombus can be set up.

Then again, a thrombus may be sterile or infected, depending upon whether or not pathogenic organisms have gained entrance into the substance of the clot.

Thrombosis, in its inception, is a protective mechanism. Nature has set up its most effective barrier in the attempt to stay the invasion of the blood stream by pathogenic microorganisms. The status, however, can not be predetermined, and the possible sequella may be one of the following:

1. *Organization: i.e.*, as a foreign body the clot stimulates connective tissue growth with capillary infiltration. As the clot becomes older the capillaries disappear and an organized connective tissue mass results.

2. *Recanalization:* Frequently the process of organization does not go to completion, but instead the thrombus becomes covered with endothelium, springing from the endothelial lining of the blood vessel, and the pressure of the circulation dilates these newly formed channels and establishes communication through the thrombus.

3. *Calcification:* In this process there is a deposition of lime salts in the thrombus. This resolves into a completely calcified mass which permanently occludes the lumen of the vessel.

4. *Suppurative Liquefaction:* When the thrombus is invaded by pus-producing organisms, suppurative liquefaction or abscess formation occurs. This process gradually progresses and eventually replaces the entire thrombus. Fragments of the infected thrombus, or the liquefied material, may be swept into the circulating blood stream and lodged in remote parts of the body, while the circulation is loaded with bacteria. This is the most unfavorable sequella of thrombosis, and it is in the presence of such a complication that the prognosis becomes grave.

The surgical management of lateral sinus thrombosis dates back to 1880, when Zaufal, while presenting a case before the Society of German Practitioners in Prague, suggested that

when sinus infection is suspected the internal jugular vein should be ligated and the sinus opened. The patient he demonstrated died from sinus empyema with basal meningitis, enlarged spleen and pneumonia. In the left sigmoid sinus there was a thrombus. In the jugular bulb and adjoining portion of the sinus, clear back to the torcular herophili, there was an old organized thrombus; the remaining portion of the sinus contained pieces of freshly broken down clot.

"It is not the thrombus," argued Zaufal, "but the breaking down of the thrombus, which causes fatality. Unfortunately, we cannot diagnose infective sinus thrombosis until after rigors and other signs, which indicate that infective particles have been carried into the blood stream, have appeared. Can we get rid of such a collection? The case I have brought forward, in which the vena jugularis on the cardiac side was occluded by uninfected clot, is like a finger post pointing the way. In such cases, I think, not only the mastoid operation but ligature of the jugular vein, and sometimes the upper part of the sinus itself, would be useful."

Zaufal reiterated his recommendations, regarding the surgical management of infective sinus thrombosis, before the same society in 1884, while presenting the autopsy specimen of a case of infective sinus thrombosis following suppurative otitis media.

Horsely, in 1886, also suggested ligation of the internal jugular vein in a case of infective sinus thrombosis, but only "directly the first indication of embolism appears."

The German otologists were skeptic about Zaufal's recommendations, and, except for discussing them at their scientific meetings, did nothing else. It was Lane, an English surgeon, who, in 1888, performed the first operation on a case of infective sinus thrombosis.

Ballance, in 1889, also performed this operation, and in his paper before the medical society of London, in March, 1890, suggested that the internal jugular vein should be tied in two places and divided between the ligatures. His reasoning was that by this method, "the upper septic portion of the jugular may be completely isolated from the lower, which remains an integral portion of the vascular mechanism."

The surgical management of sinus thrombosis gradually gained the favor of many otologists. Zaufal's recommendations, in part or in whole, were being adopted in many countries, and before long there appeared in the literature reports bearing upon the results obtained.

Viereck, in 1901, published the results of 170 operations for venous infection of otitic origin. Sixty-two cases he considered were done too late, and of the remaining 102, 89 recovered.

O. Kerner reported a series of 130 cases in which the sinus was incised and the clot removed, but the internal jugular vein was not ligated, and in which series, 77, or 58.3 per cent, recovered; while in another series of 94 cases where the vein was ligated, the number cured was 58, or 59.6 per cent.

Jones, in analyzing 50 cases of lateral sinus thrombosis, came to the conclusion that it did not matter whether or not we ligate the internal jugular vein, since the results are uninfluenced in either case.

Krepuska made a study of 295 cases of lateral sinus thrombosis. Of these, he found, 99 died and 196 recovered, and of those that recovered only 58 had the internal jugular vein ligated, while the remaining 138 recovered without ligation.

In a report of 13 cases of sinus thrombosis, Hirsch tells us that only nine recovered, and of these, five had jugular ligation, while of the four that died, three had ligation.

Gruenberg reports a series of 61 cases of sinus thrombosis in Wajatschek's clinic from 1917 to 1923, in which 20 had jugular ligation and 41 had not. In the first group, 15 per cent were cured, while in the second group 60 cures were obtained.

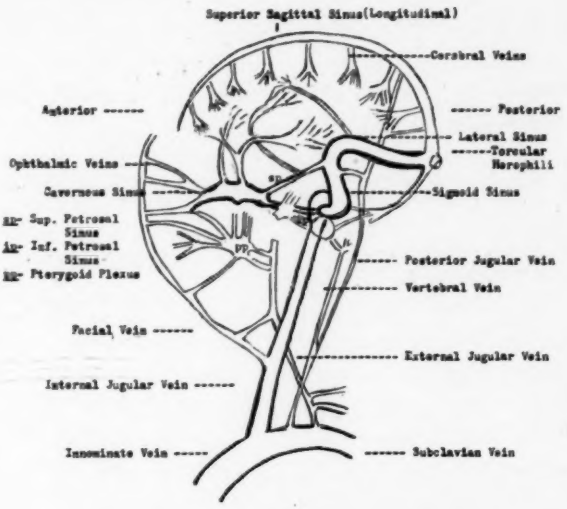
Unditz tackled this problem experimentally, producing lateral sinus thrombosis in dogs with staphylococcus, streptococcus, and a mixture of both. He divided these cases into two groups, ligated the jugulars in one group, and kept the other group for control. His results were similar in both groups (five died and six were cured), and his conclusions were as follows:

1. Ligation has no influence on the course of experimental thrombosis. *2.* Thrombosis is a protective mechanism. *3.*

Ligation is useless as a measure of closing the current, since other channels are open.

O. M. Rott is absolutely against ligation in any case of lateral sinus thrombosis, and he presents statistics and convincing arguments to back up his contentions.

From an anatomical standpoint, Dundas Grant presented several points, which, he believes, invalidate Zaufal's recommendation regarding ligation of the internal jugular vein in



Scheme of intracranial and extracranial Venous anastomosis
(According to Macewen)

a case of infective sinus thrombosis. His objection to this procedure is based on the following facts:

1. There are other channels, besides the internal jugular vein, through which the blood can reach the right side of the heart. Ligation will, therefore, bring about a reversal in the usual course of the circulation, and the blood will reach the right side of the heart through these tributaries.

2. Ligation of the jugular may cause suction of the infective material into the accessory pathways.

Gimser brought this possibility to the attention of the otologists. He observed the insuction of the wall of the lateral sinus during inspiration, after the internal jugular vein has been occluded. He concluded that ligation favors, to a large extent, the extension of septic thrombosis into the accessory pathways.

Heine published several cases in which extension by insuction has unquestionably taken place.

Dundas Grant had a case where the sinus was opened and the internal jugular ligated, yet, at autopsy, pus was found in the prevertebral veins.

Pritchard has traced the entrance of septic material into the cerebellum, through small cerebellar veins which empty into the lateral sinus.

3. Ligation of the internal jugular vein occasionally has a deleterious effect upon the brain, probably because of the difference in size that may exist between the two jugulars.

Rohrbach reported a case in which death from edema of the brain resulted following ligation of the internal jugular in an attempt to remove a tumor of the neck. The jugular of the opposite side of the neck was found too small to carry the blood to the right side of the heart.

Regarding the difference in size of the two internal jugulars, Linser has found that this exists in about 3 per cent of the cases. The left jugular is usually the smaller of the two.

In favor of ligation, on the other hand, we find such men as W. F. Wilson, of the Royal Victoria Infirmary, J. S. Fraser, Dan McKenzie, and others. Quoting McKenzie, "The resection of the jugular vein is advisable in all cases, chiefly because it is difficult to be sure that all the disease has been eradicated in any given case, and also because this detail adds but little to the severity of the operation."

The late E. B. Gleason stated that it should not be imagined that ligation is specific for sinus thrombosis. Yet he went on to say that "if the exposed sinus is covered with large, pale, flabby granulations, or is the color of dirty chamois leather, and puncture does not bring out blood, the vein should first be ligated, then the sinus laid open and the clot removed."

E. W. Day, in "Jackson and Coates," states that when the appearance of the sinus wall indicates that the thrombus is approaching the bulb, it is conservative to suppose that a mural clot or infected intima is present in the bulb and to proceed to isolate this region by ligation or resection of the internal jugular vein.

There are a great many more statistics and opinions in the literature advanced in support or proof of one procedure or the other, but to quote all these would fill many more pages and add very little to this consideration. Suffice it to say that each of the two schools has its many staunch adherents who honestly believe that their method is that of choice.

Perhaps an open-minded approach to this subject with deliberate consideration of it from all angles could serve to bring the dissenting factions together and to level the differences of opinion in regard to the management of this complication. It seems neither plausible nor scientific for any otologist to come out with a definite opinion and stand pat without keeping an open mind or ignoring suggestion. To say that in every case of sinus thrombosis, regardless of its severity or clinical picture, the vein should be ligated is as wrong as it is to insist that under no circumstances should the vein be interfered with.

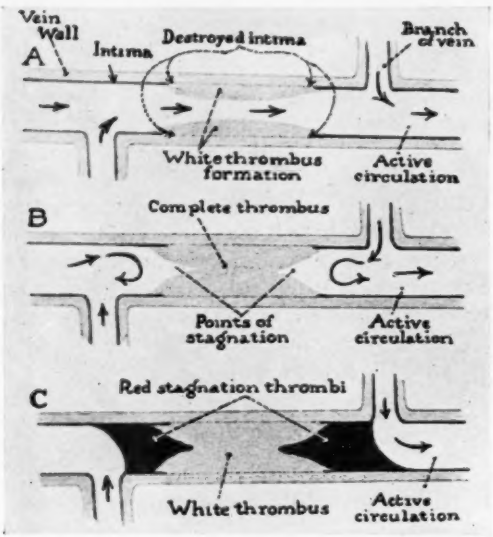
A disease presents itself in a variety of ways, and the pathology in one individual may differ in a great many respects from that in another. After all, the pathology and the clinical symptoms of a disease vary with the virulence of the organism, the resistance of the individual, and frequently with other factors; and the prognosis is entirely dependent upon these factors. Given two cases of lateral sinus thrombosis, it is not very frequent that we find the same reaction, the same clinical symptoms, or the same blood picture in the one as in the other. If all cases of sinus thrombosis would act alike, the practice of this phase of medicine would be comparatively simple and far from fascinating.

Consequently, having definitely established that we are dealing with a case of lateral sinus thrombosis, we must carefully consider the signs and symptoms as to their severity and significance, and must take into account the blood pic-

ture, including red, white, hemoglobin and differential, the blood culture, the Schilling index, and the general condition of the patient before deciding upon the course to follow.

With a given case of lateral sinus thrombosis, the method of treatment may be one of the following:

1. Medical treatment, including: *a.* Frequent small transfusions of ordinary whole blood, or blood from an immunized donor; *b.* Chemotherapy, including varying drugs; *c.* Vaccines, autogenous or stock; *d.* Sera, specific or otherwise.



2. Surgical management of the lateral sinus; *i.e.*, reopening of the mastoid wound, exposing and examining the lateral sinus, and removing the clot, or evacuating the abscess if that be found. Or, if the disease is secondary to a middle ear infection, a simple mastoidectomy should first be done, the lateral sinus exposed and examined, and the clot removed.

3. Ligation of the internal jugular vein; *i.e.*, in addition to the surgery on the lateral sinus.

As to which of these courses to follow will depend upon several factors. Thus, if the sepsis is not much more severe than it was in the primary disease, if the temperature hasn't

risen more than one or two degrees, if the Schilling index shows no shift, or only a very slight shift, to the left; if the blood culture is negative and the patient is not extremely prostrated, we are justified and safe in keeping our hands off, and watching the patient carefully. A good many of such cases have resulted in complete cure under the medical measures outlined above.

On the other hand, if the patient is not doing well under medical treatment, the temperature continues to remain high and the pulse is rapid and feeble, if the Schilling index shows an increase in the immature cells; or there is a marked and sudden drop in the leukocyte count, with evidence of increasing anemia, then it is advisable to reopen the mastoid wound or do a simple mastoidectomy if none has been done previously, expose and examine the lateral sinus and remove the clot, or drain the abscess, if such be found in or around the sinus.

Ligation of the internal jugular vein, however, should be resorted to only in selected cases. Thus, if there is a sudden increase in the severity of the toxic state of the patient evidenced by chills, very high temperature, marked prostration, accompanied by an increase in the immature cells, positive blood culture, and evidence of metastasis elsewhere in the body; or if there are indications that the infected thrombus is spreading downward toward the internal jugular vein, then ligation of the vein should be done. It will frequently help to check the spread of the infection by shutting off this channel and preventing particles of the infected thrombus and organisms from entering the circulation by this route.

The following case reports, taken from the records of the Graduate Hospital of the University of Pennsylvania, will serve to illustrate the more severe type of infection. In all of these cases thrombosis, accompanied by one or more of its unfavorable sequellae, has complicated the clinical course of the disease. Ligation of the internal jugular vein, and removal of the infected material from the sinus, was resorted to. In each case this surgical manipulation has effected a rapid, and almost instantaneous improvement in the condition of the patient, and eventually a complete recovery.

Case 102984: P. W., adult male, age 42 years, was admitted to the Graduate Hospital with the complaints of pain and purulent discharge in the left ear. The history obtained at the time of admission was

that the patient has had the "grippe" about eight weeks prior to admission. In the course of that illness his left ear became involved. Since the onset of this complication the pain in the ear has been intermittent, but the discharge has been continuous. Previous to this attack, the patient claimed, he has never had any trouble with his ears.

Examination on admission revealed a large amount of thick purulent discharge in the left ear canal. A fair-sized perforation was present in the posterosuperior quadrant of the left drum membrane. The right ear was normal, as was the rest of the physical examination. The temperature, pulse, and respiration, as well as the blood count, were within normal limits. Roentgenographic studies of the mastoids disclosed the presence of a suppurative process in the left side.

On the strength of these findings operative intervention was considered advisable, and a mastoidectomy was done. The same evening the patient had a chill, and the temperature rose to 105 degrees, at which level it remained for three consecutive days. A report of the culture made from pus found in the mastoid came back with a diagnosis of "streptococcus hemolyticus in pure culture." A blood culture,

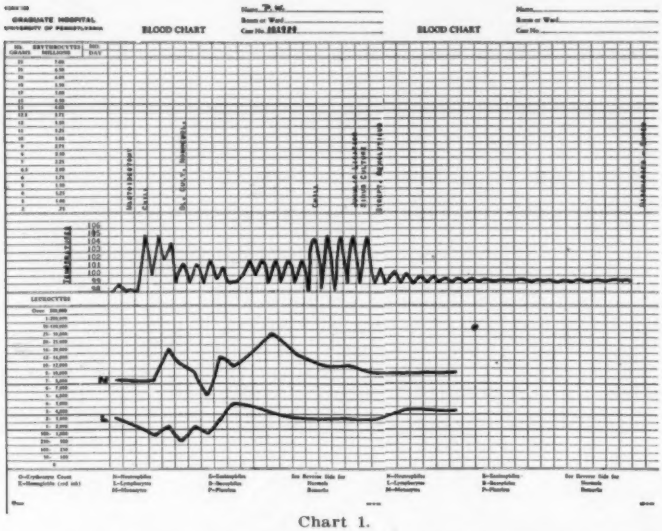


Chart 1.

taken on the second postoperative day, revealed the presence of streptococcus nonhemolyticus in the circulation. On the fourth postoperative day the temperature dropped to 102 degrees and the patient appeared much better. During the next ten days the clinical picture remained unchanged, with a daily rise of the temperature to 102. On the 14th postoperative day, however, the patient had a chill, and the temperature rose to 105 degrees. He complained of a pain in the shoulder joint. After the clinical picture remained unchanged for four days, with daily rise of the temperature to 105, and the pain in the shoulder becoming worse, exploration of the lateral sinus was thought advisable.

The old mastoid wound was reopened, and the lateral sinus was examined. The sinus was incised, and pus was found coming from the jugular end. The internal jugular vein was ligated in two places

and cut between the ligatures. The pus from the sinus was subsequently found to contain streptococcus hemolyticus in pure culture.

Two days after ligation the temperature began to drop. The shoulder joint was aspirated with the removal of 4 c.c. of pus. The patient was rapidly improving as the temperature gradually approached the normal. After a short period of convalescence in the hospital the patient made a successful recovery.

The accompanying chart gives, in detail, a clear-cut picture of the events, in chronologic order, that have taken place during this patient's illness.

Case 113773: J. H. Young, female, age six years, was admitted to the Graduate Hospital with the complaint of profuse purulent discharge from the left ear. The history obtained that the patient had gone through a siege of communicable diseases, namely, scarlet fever, chicken

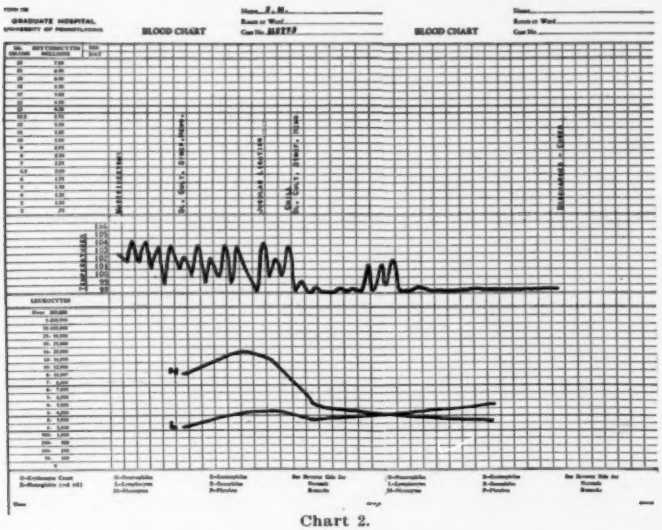


Chart 2.

pox, and measles, during the three months prior to admission. In the course of the last disease, which was measles, she developed the trouble with her left ear. Her chief complaint at the time was pain in the ear. Examination revealed an acutely inflamed and bulging ear drum. Incision of the latter brought relief from pain and abatement of the other symptoms. Ten days after myringotomy, however, her ear condition became worse. She developed a high fever and complained a good deal of headache. The ear began to discharge profusely.

On admission the patient appeared toxic, and complained of headache. The temperature, pulse, and respiration were 103, 104 and 28, respectively. The left ear was filled with a thick purulent material, and there was a large perforation in the anterior portion of the drum. The left mastoid was red, swollen and extremely tender. The rest of the examination was negative.

In view of the severity of the condition a mastoidectomy was decided upon. The mastoid was found badly diseased, with necrotic cells at

the tip and around the lateral sinus. The immediate postoperative reaction was good, but the temperature rose to 104 and the patient became toxic. Pediatric consultation failed to disclose any systemic condition that might account for the sudden change in the clinical picture. A blood culture, taken at the time, disclosed the presence of streptococcus hemolyticus in the circulation. Antistreptococcus serum and Pregl's iodine were administered, but without success.

With all other therapeutic measures being of no avail, ligation of the internal jugular vein was done. The mastoid wound was reopened and the lateral sinus exposed. Packing was inserted at both ends of the sigmoid sinus. Upon incising the latter, clots and pus were found and removed. Removing the packing, one at a time, revealed bleeding from above and none from below. The internal jugular vein was then ligated in two places above the common facial vein and left intact.

For two days following the operation the temperature remained at 104. An immuno blood transfusion of 300 c.c. was given, and this was followed by a chill and a drop in the temperature to 99 degrees. Thereafter the condition gradually improved and, with the exception of a few days' rise, the temperature remained normal and the child recovered completely.

The above case report, in summary, presents the following salient features: 1. A virulent mastoid infection. 2 A prolonged high temperature following mastoidectomy. 3. Repeated blood cultures positive for streptococcus hemolyticus. 4. Thrombi and purulent material in the sinus. 5. Absence of bleeding from the lower end of the sinus.

In the presence of these features, and with the failure of other therapeutic measures, ligation of the internal jugular vein is justifiable and, in fact, commendable. It frequently saves the life of the patient, and, therefore, should be done.

The accompanying chart presents, in detail, the essential features of this case.

Case 104770: K. B., adult male, age 44 years, came into the hospital with the complaint of purulent discharge from the right ear and soreness over the mastoid on the same side. The history obtained was that he had contracted cold about two weeks previous to admission. Two days after onset he had experienced soreness behind the right ear. He had also noticed that his hearing in that ear was becoming impaired. Elevation of temperature has been present since the onset. The physician in attendance found the ear drum bulging and he incised it. Since then the ear has been continuously discharging a thick, purulent material.

Examination, on admission, revealed the presence of a thick purulent discharge in the right ear. The mastoid tip was quite tender, but did not show any external evidence of involvement. The tonsils were large, cryptic and diseased. There was marked cervical adenopathy. The temperature, pulse and respiration were 98, 112, and 22, respectively.

In view of the above findings a diagnosis of acute mastoiditis was made, and a mastoidectomy was performed. In the course of the operation the lateral sinus was accidentally injured.

The immediate reaction to the operation was good, and the patient presented no complaints. On the third postoperative day, however, the patient had a chill, and the temperature, pulse, and respiration rose to 104.2, 130, and 40, respectively. A blood count, taken at the time, came back with a report of 14,000 leukocytes and 91 per cent neutrophiles. During the next ten days the temperature kept on fluctuating between 100 and 103, with the pulse correspondingly high and the respirations around 30. On two consecutive days, in this period, the temperature

rose to 104. The nuclear index showed a variation from 6.6 and 14.5 The patient was quite toxic and apprehensive about his condition. A blood culture taken during this period came back with a report of staphylococcus albus.

On the 16th postoperative day the patient began to complain of pain in his knee. This was considered manifest of metastasis, and ligation of the internal jugular vein was considered imperative. The old mastoid wound was reopened and the sinus was fully exposed. Incision of the latter revealed the presence of a large amount of pus, which was subsequently reported to contain staphylococcus albus. The jugular vein was then ligated.

Following the ligation the patient's condition began to improve steadily. The temperature dropped gradually, the nuclear index returned to normal, and the pain in the knee completely disappeared. Within ten

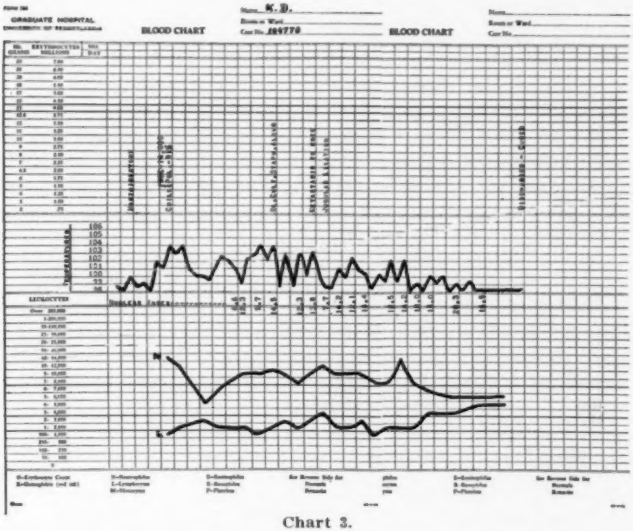


Chart 3.

days after ligation the patient was discharged from the hospital in good condition.

The accompanying chart points out clearly the salient features of this case. The continuously elevated temperature, the chill, positive blood culture, and metastasis to the knee point to a very severe infection. One thing, in particular, i.e., the nuclear index, is of interest in this case. It will be noted that the nuclear index followed the course of the disease rather closely, and that after ligation, it returned to normal and remained there till the day the patient was discharged from the hospital.

The argument has often been raised that shutting off of this vessel does not assure the prevention of the spread of the infection to the main circulation. This is very true, since, as you will recall from the anatomical consideration, there

are accessory pathways through which the infection can reach the venous side of the heart; but by ligating the jugular vein, we at least block off the main thoroughfare for the infected emboli.

Some may similarly argue that ligation is of no value since we often find that even after ligation symptoms of metastasis occur, but we must not lose sight of the fact that these symptoms may be due to a secondary focus established before ligation of the internal jugular vein has been undertaken.

In conclusion, let me repeat that in a given case of lateral sinus thrombosis, definitely established with the aid of all diagnostic means at our disposal, one should not act hastily. With very few exceptions, this is not an emergency where one has to rush in and ligate the jugular immediately. The patient should be carefully observed and treated medically as long as his or her condition permits, and just as soon as the surgeon feels that more radical measures are essential he should reopen the mastoid wound, or do a mastoidectomy if such has not been done, then plug the sinus above and below, incise it and test for bleeding from each end. Now, if there is bleeding from both ends and we are reasonably sure that the blood is coming from the sinus itself and not from its tributaries, and we may then assume that there is no thrombosis in any part of the sinus or the jugular vein, the sinus should be packed, the mastoid incision dressed, and nothing further be done.

If, however, there is bleeding from below and none from above, we may assume that there is a thrombus somewhere between the upper plug and the torcular herophili. The thrombus should be looked for and removed, if possible; the sinus packed and the mastoid wound dressed. Ligation of the internal jugular vein in this instance will be of no further help since in this area there are many other pathways for the infection to gain entrance to the systemic circulation. On the other hand, if bleeding is obtained from above, and none from below, which means that a thrombus is in all probability present either in the bulb or the vein itself, then ligation of the internal jugular vein, followed by removal of the clot, is justified and should be done.

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1726 Eye Street, N.W.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Announcement is herewith made by the American Academy of Ophthalmology and Otolaryngology of a change in the 1937 convention site to Chicago, Oct. 10 to 15, 1937; headquarters, Palmer House.

It is with deepest disappointment, the officers, with the approval of the Council, have, because of mechanical difficulties, found it necessary to change the meeting place from Detroit to Chicago.

The Society regrets circumstances developed to prevent honoring the Detroit members by taking the 1937 session to their home city.

PETROUS APEX SUPPURATION INVOLVING THE CAROTID CANAL AND CAUSING A HORNER'S SYNDROME.*†

DR. MERVIN C. MYERSON, New York.

Infections which spread from the middle ear to the petrous pyramid may localize in either the upper or lower part of the apex area. Those which localize in the inferior portion of the apex will involve the bone surrounding the internal carotid artery. The canal which lodges the internal carotid artery in the petrous bone measures 1½ to 2 cm. in length and is anteroinferiorly placed in its relation to the apex cavity. When the infection involves the sympathetic nerve plexus surrounding the artery, a Horner's syndrome results. The pupil of the eye on the same side is contracted, the palpebral fissure is narrowed, and the size of the eye appears to be smaller. Stacy Guild,[1] discussing the symptomatology of petrous disease from the anatomic and physiologic standpoint, mentioned this syndrome as a possibility. Up to the present time a case of petrous pyramid suppuration presenting this syndrome has not been reported.

The patient complained of pain deep in the glenoid fossa upon moving his jaw. This symptom has not been mentioned in connection with petrous pyramid infection.

The clinical picture was confused by the presence of chronic suppurative sphenoethmoid disease, and a concentric contracture of both visual fields. This led us to perform a sphenoethmoidectomy. A few days before he developed signs of meningeal irritation; the labyrinth on the other side was found to be dead.

The area of the carotid canal and the apex were successfully drained through a cervical incision.

Case Report: A white man, age 44 years, was first seen by us on Feb. 1 of this year. He had been admitted to

*Read before the New York Academy of Medicine, Section on Otology, May 21, 1937.

†Department of Otolaryngology, Kings County Hospital.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 7, 1937.

the Otolaryngological Service for surgery of the sinuses on Nov. 2, 1936. For many years he had suffered with headaches and was subject to frequent sore throats. X-ray and clinical study revealed the presence of bilateral pansinusitis with more marked involvement of both maxillary sinuses. On the day of admission a Caldwell-Luc operation was performed upon the left antrum.

His postoperative course was uneventful until the seventh day, when he developed an acute upper respiratory infection. Six days later he complained of pain and fulness in both ears, subsequently developing suppuration in both middle ears, so that on the twenty-first the ear drums were incised. His mastoid processes appeared to be involved simultaneously with the middle ear cavities. On Dec. 7, it became necessary to perform a simple mastoidectomy on the left temporal bone. At operation a perisinus abscess was found; there was no indication that a thrombus was present in the sinus.

The right ear became dry and remained so for some days. On Dec. 22, he complained of headache and dizziness. A rotary nystagmus directed to the left side was noted. Seven days later the right ear again discharged. The discharge continued. A few days later tenderness appeared over the right mastoid area and he complained of severe right temporal headache. On Jan. 4, a horizontal nystagmus to both sides, more pronounced to the left, was noted. Hearing in both ears was good and both labyrinths proved to be functioning normally according to the caloric tests. The spinal fluid was removed under slight pressure. Examination revealed 30 cells, mostly lymphocytes, in each cubic millimeter.

On the next day a mastoidectomy was performed; extensive destruction of bone was found. The inner cortex in the region of the sinodural angle was found destroyed and granulations were seen on the cerebellar dura. A good portion of the plate of bone covering the sinus was removed, revealing a normal appearing sinus wall.

The postoperative condition was satisfactory, but the patient continued to complain of pain in the temporal region. On Jan. 22, despite the return of his temperature to normal, he complained of right frontal, temporal and occipital headache.

He came under our care on Feb. 1. At this time the general physical examination was essentially negative; the neurological examination was also negative. The optic nerve-heads showed no change from normal; X-ray studies of the petrous pyramid were negative. Examination of the nasal passages with the Holmes nasopharyngoscope revealed small polypi in the right sphenoethmoid region, with a small amount of pus coming into this area, apparently from the sphenoid.

Careful questioning of the patient and his family revealed that he had suffered with severe headache in the frontal, temporal and occipital regions for several years. On Feb. 11, it was noticed that the right pupil was smaller than the left, that the palpebral fissure was definitely narrower than normal and that there was an apparent shrinkage of

Fig. 1. Photograph of eyes to show Horner's syndrome on right side. The pupil is contracted, the palpebral fissure is narrowed and the eye is apparently diminished in size.

the size of the eyeball. These signs were interpreted as a Horner's syndrome (see Fig. 1). X-ray studies of the petrous area were repeated, but were not significant.

On Feb. 12, the ophthalmologist reported the presence of a concentric contracture of both visual fields, more pronounced on the right side. Because of this and the presence of nasal sinus disease, a right intranasal sphenoethmoidectomy was performed. Some polypoid tissue and a small amount of pus was encountered in the sphenoid and the posterior ethmoid region. Following the operation the headache was relieved, but for only two days. The visual fields returned to normal by Feb. 25.

Neurosurgical examination was negative except for the signs pointing to the Horner's syndrome. On March 2, the

neurologist reported a suggestion of a nystagmus to the right, but this was not corroborated. Two days later the patient complained of deep-seated pain in the right glenoid fossa which was present on opening and closing his mouth. On this day, for the first time, the X-ray film indicated involvement of the inferior aspect of the petrous apex. In the film taken in the Stenver position the line of the roof of the apex was intact, while in the film taken in the base or mento-vertical position, there was a loss of definition of the apical structure (see Figs. 2 and 3). These diagnostic Roentgen signs, together with the deep-seated pain in the glenoid fossa and the Horner's syndrome, indicated an osteomyelitis of the inferior aspect of the petrous pyramid.

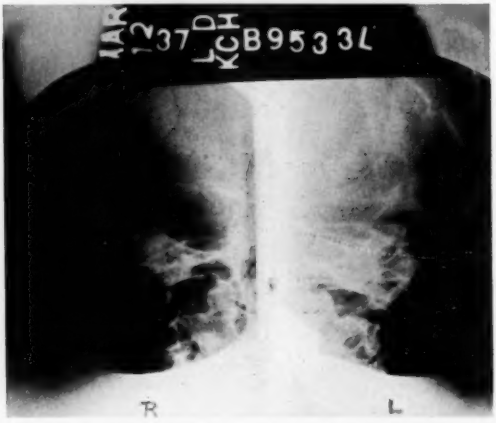

Fig. 2. Roentgenogram of petrous pyramid in Stenver position. The line of the roof of the apex is intact.

On March 6, we explored this area by means of the cervical approach recently published.[2] The retropharyngeal space was entered by means of an anterior sternomastoid incision. Upon reaching the roof of this space the pharyngeal aponeurosis was penetrated and softened bone was encountered. An angular probe was gently placed through the softened bone. Its withdrawal was followed by a gush of approximately three drams (12 cc.) of pus. A drain was inserted deep into the recess and the wound closed, except at the lower

angle. On the day following the operation the patient was entirely free from headache.

On the seventh day the middle ear was completely dry, but the temporal headache had returned. On the next day the headache was severe as before the operation. Frontal and occipital headache, however, were no longer present. On the eighth day (March 14), it was noticed that he could not hear with his left ear. Examination revealed a dead cochlea and a dead labyrinth. The Horner's syndrome re-

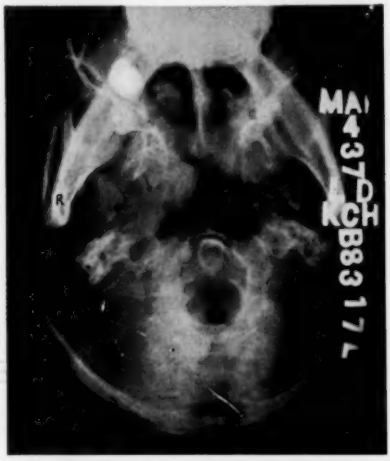

Fig. 3. Roentgenogram in the base or mentovertical position. The definition of outline of bony septa in the apex area is lost.

mained unchanged. Two days later the spinal fluid was removed under slight pressure. It contained 70 cells per cubic millimeter.

The temporal surface of the petrosa was now explored. An irregularly circular erosion was found directly above the internal auditory meatus. From the medial boundary of this eroded area a small opening was found leading into the apex cavity. There was no pus in the apex cavity. Some granulations were found on the dura adjacent to the area of erosion. The question of whether this small area might lead into a subdural or cerebral abscess was considered. The

headache was greatly relieved, but recurred on the next day. Three days later the patient became stuporous; he presented a mixture of neurological signs indicative of a spreading meningitis. His spinal fluid now contained 2,500 cells per cubic millimeter, of which 85% were polymorphonuclear. Streptococci were seen on smears. They proved to be hemolytic when studied in cultures. The temporal lobe was again elevated and the previously diseased area of dura exposed. This was incised but yielded nothing; likewise incision into the basal cistern was followed neither by pus nor spinal fluid. The patient expired the next day. An autopsy could not be obtained.

The following features of this case are mentioned:

1. On Dec. 22, a nystagmus toward the left was noted, which indicated a perilabyrinthine spread of the infection.

2. A concentric contracture of the visual fields was present which led to the performance of a sphenoethmoidectomy.

3. A Horner's syndrome was present.

4. Deep-seated pain in the glenoid fossa occurred on moving the condyle of the mandible.

5. The apex and carotid canal area were successfully drained by means of a cervical incision.

6. The labyrinth on the opposite side was found to be dead two days before a spinal fluid cell count of 70 appeared.

7. An erosion of the anterior surface directly above the internal auditory meatus was found leading into the apex cavity.

8. Neither pus nor spinal fluid were found on incision of the dura of the temporal lobe or over the basal cistern.

Did the meningitis spread from the area of involved dura, or was there an unrecognized brain abscess? It is frequently very difficult to diagnose the premeningitic phase of a brain abscess. It is doubtful whether a suppurative labyrinthitis of the other side caused the terminal meningitis, yet this must be considered as a possibility.

A case of petrous pyramid infection with involvement of the apex and the carotid canal area, is reported. A Horner's

syndrome resulted from the carotid lesion. The infection originated in the inferior portion of the apex area, later spreading to the anterior surface directly above the internal auditory meatus.

Earlier exposure of the temporal surface of the bone might have served us better in this case.

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136 East 46th Street.

FOURTH INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS.

The fourth International Congress of Oto-Rhino-Laryngology will be held in Amsterdam in 1940. The week from July 29 to Aug. 3 has provisionally been settled for the Congress.

A Committee of Honour has been appointed, composed of the professors of oto-laryngology in the Netherlands: C. E. Benjamins, Groningen; H. Burger, Amsterdam; P. H. G. van Gilse, Leiden; P. Th. L. Kan, Wassenaar; A. de Kleyn, Amsterdam; F. H. Quix, Utrecht; W. Schutter, Groningen; H. J. L. Struycken, Breda.

The Bureau of the Congress consists of the following members: Prof. H. Burger, Keizersgracht 317, Amsterdam C., President; Dr. A. A. J. H. Marres, Willemsparkweg 31, Amsterdam Z., Hon. Secretary; Dr. M. J. ten Cate, Obrechtstraat 55, Amsterdam Z., Hon. Treasurer.

HEMANGIOMA OF THE SOFT PALATE. CASE REPORT.

DR. BEN L. BRYANT, Los Angeles.

In his volume on Neo-plastic Diseases, Ewing explains the origin of the hemangiomata by referring to them as a developmental anomaly in the structure of certain vascular segments which do not fit into the circulatory system and which retain embryonal characters. He does not mention the soft palate as a site of such tumor, nor does Furstenberg in his treatise on Tumors of the Nose, Pharynx and Mouth, although the latter does note that they are occasionally found in the lips, tongue, buccal mucosae and in the mucous membranes of the nose. Furstenberg further states that Ribbert found that the simple hemangioma develops in a portion of a blood vessel wall and from that point produces a proliferation of newly formed vessels which invade the neighboring tissue but do not connect with any of the normal blood vessels in the surrounding structures. The treatment of this condition which has been advocated by various writers includes surgery, cauterization, electrocoagulation and radium, with the decision to be made in a specific case as to which method is desirable and applicable.

The following case is reported because of the location of the lesion, its growth, and the effect of one radium treatment:

Case Report: M. F., white, female, age 23 years, was examined on Aug. 11, 1932. There was a slightly perceptible scar on the left cheek in the parotid region, where a year before a tumor had been excised which was reported to have been a hemangioma. It was about the size of a bird's egg and had been present for a number of years. Examination of the mouth showed that the left side of the soft palate was normal in appearance. The right side presented a swelling about the size of an olive, purplish in color, with a small superficial area of redness in the center. This was diagnosed as a hemangioma and the patient was advised to return in several weeks for further observation.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 8, 1937.

She did not reappear, however, until March 9, 1936. At this time she complained of a constant impulse to swallow, particularly when reclining. Examination revealed that the tumor in the right side of the soft palate had increased to the size of a large plum, extending backward to the posterior tonsillar pole, medially to the midline of the palate, and forward to the last molar. On palpation, the mass was found to have the consistency of a rubber sponge, and pulsation could be felt.

A Roentgenologist was called in consultation, and on the next day treated the patient as follows: 75 mg. of radium contained in six steel needles (each needle containing 12½ mg. of radium) were placed on a wooden tongue blade and spaced so as to cover the entire tumor. The steel needles were covered only by two layers of dental rubber dam and the tongue blade was held against the lesion for two hours.

The patient was seen on March 28, 18 days after the treatment. The tumor had completely disappeared, with only a small area of induration remaining on the right side of the soft palate. On April 18, the area of induration had almost disappeared. On June 6, nothing abnormal could be determined in the previously affected area; and on Jan. 5, 1937, the region was completely normal in appearance and, of course, the patient was without complaint.

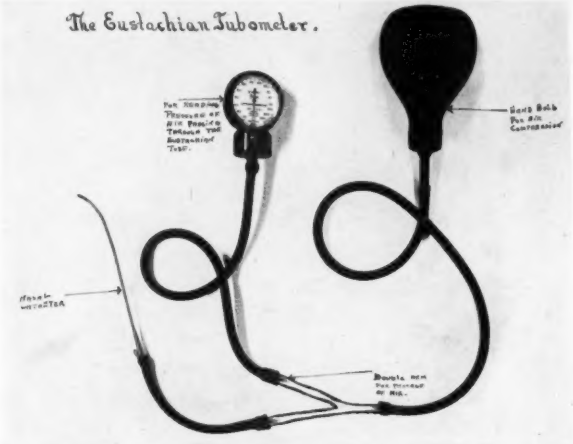
Comment: It is probable that the tumor described in this report was a metastasis from that which had been present in the left parotid area for years, and which had been removed a year previous to the first examination reported above. The complete and rapid disappearance of this tumor following one radium treatment indicated its radiosensitivity, and it was fortunate in this instance, because it was possible to avoid the mutilation and possible disturbance of function which would probably have followed any other form of treatment.

2007 Wilshire Boulevard.

A NEW DEVICE—THE EUSTACHIAN TUBOMETER.

DR. M. MARTYN KAFKA, Brooklyn.

There are many occasions in the practice of the otologist where there is a desire to know how much of the opening is present in the Eustachian tube. Such information is of great value, particularly in the treatment of the middle ear diseases, such as adhesions of the ossicles in the middle ear, pus in the middle ear, chronic purulent otitis media, obstructions of the Eustachian tube, hypertrophic lymph tissue in the orifice of the Eustachian tube and its canals, etc.



The majority of the books give the length of Eustachian tube as 36 mm. and the direction of the tube as downward, medialward and forward; the angle in this case equals 45° with the sagittal plane, and the other angle as 30 to 40° with the horizontal plane. The length of the bony Eustachian tube is 12 mm. long. The cartilaginous part of the Eustachian tube is 24 mm. long. I have written to the Museum of Natural

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication. April 24, 1937.

History for information of the general diameter of the human Eustachian tube, and have searched in textbooks of anatomy and otology, but I have failed to obtain any definite information, either from the Museum of Natural History or the textbooks.

I have, after careful experimentation, come to the conclusion that the average diameter of the cartilaginous human Eustachian tube is about 2 to 2.25 mm. and I have found that

the bony diameter of the average human Eustachian tube is 1 to 1.25 mm., and pressure obtained with the Eustachian tubometer was from 260 mm. to 1.50 mm.

With careful experiments which I carried out at different intervals and with the use of various types of tubes having graded diameters of the orifices, I obtained the following results. I used the Eustachian tubometer for readings thus: With the complete occlusion of aperture, reading with instru-

ment was 340 mm. pressure. When the diameter of tube used was 1.25 mm. the average pressure obtained was 240 mm. On using another tube with diameter of 2 mm., the general average pressure reading was 170 mm. With the employment of a tubing with a 3.5 mm. diameter, the pressure obtained was 60 mm. On checking the tubing with the diameter of 5 mm. the general resultant reading was 30 mm., and when a piece of tubing which diameter was 6 mm. the general average reading was 10 mm. Charted thus:

Diameter Complete closure	Pressure Reading
1.25 mm.	340 mm.
2.00 mm.	240 mm.
3.50 mm.	170 mm.
5.00 mm.	60 mm.
6.00 mm	30 mm.
	10 mm.

By meticulous observation and study I have found that this device will greatly accelerate the speed in observing the desired information relative to the therapy and deafness in the middle ear diseases.

One can readily see that with the variations in diameters and with obstructions in the orifice in the cartilaginous and bony canals of the Eustachian tubes that the air passing through these canals will register different pressures procured through various types of canals.

807 St. Marks Avenue.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of April 21, 1937.

SYMPOSIUM ON BACTERIAL MENINGITIS.

The Clinical Picture of Bacterial Meningitis with Particular Reference to Its Changing Phases. Dr. Samuel J. Kopetzky.

(Published in full in this issue.)

The Pathways of Infection:

1. **The Paranasal Sinuses.** Dr. Rudolph Kramer.
2. **The Ear.** Dr. Marvin Jones.

(Published in full in this issue.)

Differential Diagnosis of Otitic (Bacterial) Meningitis. Dr. James G. Dwyer.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Differential Diagnosis of Suppurative Meningitis Caused by Paranasal Sinus Disease, with Some Suggested Prophylactic Measures. Dr. Charles J. Imperatori.

(Published in full in this issue.)

Differential Diagnosis of Extraotitic Meningitis. Dr. E. D. Friedman.

(Published in full in this issue.)

A Summary of Methods Used in Treating Meningitis Secondary to Infections of the Ears and Sinuses. Drs. Josephine B. Neal, Henry W. Jackson and Emanuel Appelbaum.

(Published in full in this issue.)

The New York Meningitis Committee of the American Otological Society. Dr. Thos. J. Harris.

(Published in full in this issue.)

DISCUSSION.

DR. ANDREW A. EGGSTON: I had occasion some years ago to review the deaths at the Manhattan Eye and Ear Hospital from 1926 to 1934—a period of eight years. There were 363 deaths in the institution during that time, and 48.5% died of intracranial suppurative disease.

Total deaths for eight-year period between 1926-1934 363

Deaths from Intracranial Suppurative Disease.

1. Meningitis	110
2. Meningitis and Brain Abscess	53
3. Brain Abscess	14
Total	177, or 48.5%

Pathways of Suppurative Meningitis.

1. Auditory Apparatus	90
2. Paranasal Sinuses	18
3. Cavernous Sinuses	8
4. Postoperative	8
5. Miscellaneous	3
Total	124, or 70%

Pathways of Brain Abscess.

1. Auditory Apparatus	37
2. Paranasal Sinuses	18
3. Osteomyelitis, etc.	3
4. Miscellaneous	9
Total	67, or 30%

No doubt these statistics can be repeated in other hospitals all over the country. This tragical situation certainly places the specialty of otolaryngology in a major position in medicine, although unfortunately it has not received the attention it deserves in medical teaching. Furthermore, autopsies which are performed on these cases in most institutions have been very perfunctory. Very little information is gained if only it is decided that a patient has a brain abscess or meningitis. Of most importance is to learn the pathway by which the infection entered the cranial cavity. The only way in which to understand clearly the mode of infection and the method of treatment is by autopsy exploration of the bones of the nasal accessory sinuses and of the petrous portion of the temporal bone, followed by careful microscopical examinations of serial sections of these structures.

It therefore behooves every one of the members of this Section, if they have a death from intracranial suppurative disease, to insist that the pathological department conduct a detailed study of the bony vault. This Section should by all manner of means encourage a more determined study of the fundamentals of chemistry, physiology, histology, and pathology as related to the structures involved in otorhinolaryngology. Nothing will do more to place the specialty upon a better and more scientific basis. Such a program is in line with the adopted policies of other and better appreciated specialties in medicine.

Dr. Wesley C. Bowers: After these very comprehensive papers this evening, there is little left for me to add. However, I should like to stress a couple of important points. The symptoms of meningeal irritation which are frequently overlooked are: *1.* photophobia, and *2.* pain (not rigidity) in the back of the neck on flexing it upon the chest. When combined with a high blood count, they mean to me that there is certainly meningeal irritation. When coupled with increased pressure or increased cell count in the spinal fluid, they call for a very thorough removal of the underlying focus of infection. In looking for such a focus in the mastoid bone, a good job has not been done until all the bone over the mastoid portion of the posterior fossa has been removed. Sometimes an abscess on the posterior surface of the petrous bone may be thus discovered. Likewise, the cells in front of the sinus, between it and the facial nerve, must be followed to the limit, as at this point an abscess on the inferior surface of the petrous bone may be

revealed. Next, the bone over the mastoid portion of the middle fossa must be removed and all cells around the semicircular canal thoroughly investigated for a possible abscess on the superior surface of the petrous bone. If all these points fail to reveal any source of existing meningeal irritation, then I feel that a radical operation should be done, the drum and the ossicles removed and the search continued down around the Eustachian tube and below the promontory.

Too often, when we have meningeal symptoms and are fearful of meningitis, insufficient surgery is done to eliminate entirely all foci of infection.

Dr. Samuel J. Kopetzky: I doubt whether it is because of a lesser virulence that we are seeing more recoveries than we did before. What produces these cures must be evaluated. We are getting a clinical picture now that we do not understand. Rarely had we cases that ran from 10 weeks to eight months. In one I saw that went to autopsy after a long time, where the fluid became sterile and clear, there was found a pyogenic membrane in the ventricles which shut off the fluid and formed the hydrocephalus. Secondly, we now have cases in which the patients have all the evidence of an encephalitis reaction, showing the same clinical picture as postencephalitic cases, with no temperature and a gradual resumption of cerebration and all its functions. These are cases that are beginning to come as a result of our better therapy. The prognosis as to pneumococcus meningitis and otitis meningitis is very different. It is better in otitic meningitis because surgery is more easily applicable in the removal of foci than in the other type.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION ON OTO-LARYNGOLOGY.

Meeting of March 12, 1937.

DR. LAWRENCE R. BOIES gave a paper on "Extradural Inflammation: A Study of Its Occurrence in Acute Surgical Mastoiditis." Lantern slides were shown.

The writer has often been impressed, when doing a mastoidectomy, with the frequency with which the inflammatory process has actually reached the dura over the lateral sinus or at the middle fossa. He has also been impressed with the probability that certain anatomical factors in the mastoid influenced the development of an extradural inflammation at a particular date. This study was made to reduce these impressions to actual facts. It was reported at the New York meeting of the Academy and those who are interested in the complete paper may read it in the Transactions of the Academy for 1936. At this time the author simply summarizes his observations as follows:

A study of 300 consecutive cases of acute surgical mastoiditis occurring over a five-year period and with a normal age distribution indicates that inflammation actually reached the dural covering of the lateral sinus or the middle fossa or along the surface of the petrous pyramid in 35 per cent of the cases. The involvement was at the lateral sinus in 21.6%, and over the middle fossa in 5.6%. Both lateral sinus and middle fossa were involved in an additional 5.6%. There was evidence that the dura along the surface of the petrous pyramid was involved in 2.3% of the cases.

An anatomic factor was observed in a limited number of cases to be an apparent influence in the development of the inflammation at the particular site noted. This observation was limited to those cases with a relatively short duration of the otitis in which there was not a diffuse involvement of the mastoid structure, but rather a localized area of maximum destruction, and the presence of some unusual anatomic arrangement at this site.

The incidence of inflammation reaching the lateral sinus seemed to be influenced in these cases by the following anatomic factors, in order of frequency: 1. An abnormally forward position of the lateral sinus; 2. Extensive development of posterosuperior angle cells; 3. Large cells at the tip coming into close relationship to the lateral sinus.

The anatomic influence to inflammation reaching the middle fossa seemed influenced chiefly by extensive pneumatization in the zygoma or squama, or both, and to a less extent by a low tegmen.

The high incidence of extradural inflammation emphasizes the need of a complete mastoidectomy. In this series this required, in over one-half the cases, an exposure of the dural covering of the lateral sinus or of the middle fossa or both.

The mortality in this series was four per cent, including three instances in which the patients were admitted in a condition beyond reasonable hope of surgical aid.

DISCUSSION.

DR. HORACE NEWHART said he was sorry the essayist had not taken time to give more details of his paper, for he considered it one of the most

instructive papers that had been presented for a number of years. Much of the material was from the Division of Otolaryngology of the University of Minnesota.

The facts brought out emphasize the necessity of more thorough work in mastoid surgery. In past years, otologists have operated patients with acute mastoiditis a second and even a third time, or, exceptionally more often, in order to relieve the symptoms. In recent years, otologists have come to regard the "complete" operation rather than the "simple" mastoid operation as the one of choice in acute cases. The former involves cleaning out all accessible mastoid cells which by any chance may be or may become infected, exposing the healthy bone directly overlying the sinus and the dura of the middle fossa. When the findings do not account for the symptoms, the sinus and/or the dura, or both, should be exposed for inspection. When this procedure becomes the rule our mortality from the complications of middle ear suppuration will be materially reduced.

Dr. H. L. Williams felt there was not much to be added to Dr. Newhart's discussion of this paper. A complete mastoidectomy has been the end to be sought since Küster established the basic principles of mastoid surgery in 1889. Dr. Williams was sure the more complete the mastoidectomy, the more perfect the results would be.

Dr. M. W. Wheeler said he had found that if he took a little more time and cleaned out the cells completely, the time of healing was cut much shorter than if he did not. On numerous occasions it has been necessary to go back into the operative field and usually there is a large mass of cells not outlined distinctly and particularly in the tip region. Purely from the standpoint of not liking to work, it is more important to clean out all the cells demonstrable with X-ray. When that is done, it is not unusual to see them heal in two or three weeks and they do not have to be dressed indefinitely.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Fifty-ninth Annual Congress of the American Laryngological Association was held at the Marlborough-Blenheim, Atlantic City, N. J., on May 31, June 1 and 2, 1937, under the presidency of Dr. William B. Chamberlin. Prof. R. H. Stetson, of Oberlin College, was guest speaker.

It is with deep regret that the secretary announces the following deaths: Dr. John S. Fraser, Edinburgh, Scotland, Corresponding Fellow, May 11, 1936; Dr. Frederick C. Cobb, Bradenton, Fla., Emeritus Fellow, Oct. 12, 1936; Dr. Henry L. Wagner, San Francisco, Cal., Emeritus Fellow, Dec. 27, 1936; Dr. Robert C. Myles, New York, N. Y., Emeritus Fellow, Jan. 1, 1937.

Dr. F. R. Nager, of Zurich, Switzerland, was elected to Honorary Fellowship; Dr. Georges Portmann, of Bordeaux, France, to Corresponding Fellowship; Dr. Harry A. Barnes, of Dedham, Mass., to Emeritus Fellowship; and the following to Active Fellowship: Dr. Robert E. Buckley, 50 East 63rd street, New York, N. Y.; Dr. Samuel James Crowe, 4332 North Charles street, Baltimore, Md.; Dr. Carlton Stewart Nash, Medical Arts building, Rochester, N. Y.; Dr. George L. Tobey, Jr., 270 Commonwealth avenue, Boston, Mass.

The following officers were elected: President, Dr. John F. Barnhill; 1st Vice-President, Dr. Frank Robert Spencer; 2nd Vice-President, Dr. Bernard J. McMahon; Secretary, Dr. James A. Babbitt; Treasurer, Dr. Charles J. Imperatori; Librarian and Historian, Dr. Geo. M. Coates; 1st Councillor, Dr. Horace Newhart; 2nd Councillor, Dr. Dunbar Roy; 3rd Councillor, Dr. B. R. Shurly; 4th Councillor, Dr. Wm. B. Chamberlin.

Will all Fellows desiring to present papers at the 60th Annual Congress communicate promptly with the secretary, Dr. Jas. A. Babbitt, 1812 Spruce street, Philadelphia?

AMERICAN OTOLOGICAL SOCIETY, INC.

The seventieth annual meeting of the American Otological Society was held under the presidency of Dr. Edmund Prince Fowler, of New York, at the Lido Country Club, Long Beach, Long Island, N. Y., May 27 and 28, 1937. There were present 66 Active, three Senior and seven Honorary members, as well as 75 guests. The program of the meeting was devoted largely to an exhaustive discussion of Nerve Deafness considered in all its various aspects and called forth general praise for the excellency and completeness of its presentation.

Upon recommendation of the Council, the following were elected active members:

Dr. Charles Edward Connor, of St. Paul; Dr. Austin Albert Hayden, of Chicago; Dr. Samuel Rosen, of New York; Dr. William P. Wherry, of Omaha; Dr. Henry Marks Goodyear, of Cincinnati; Dr. Robert L. Moorhead, of Brooklyn; Dr. Le Roy Allan Schall, of Boston. Dr. F. R. Nager, of Zurich, Switzerland, was elected to Honorary membership.

Dr. Harris P. Mosher, Dr. Isidore Friesner, Dr. Thomas J. Harris, Dr. Francis R. Packard, Dr. Edmund Prince Fowler, Dr. Horace Newhart and Dr. D. E. Staunton Wishart were elected members of the Council for 1937-38.

The Council elected as officers for the ensuing year, Dr. Harris P. Mosher, President; Dr. Isidore Friesner, Vice-President, and Dr. Thomas J. Harris, Secretary-Treasurer.

The Society has lost by death during the past year three Senior members: Dr. Robert C. Myles, died Jan. 1, 1937; Dr. Philip Hammond, died Feb. 7, 1937; Dr. Franklin M. Stephens, died May 20, 1937.

The New York and the Chicago Committees on Otitic Meningitis made reports of the study of that subject being carried on by these two committees.

The next annual meeting will be held in May, 1938, in Atlantic City, as a constituent member of the Congress of American Physicians and Surgeons, which meets at that time and place.

You are earnestly requested, if you have not done so, to forward to the Secretary, Dr. Thomas J. Harris, the Biographical Questionnaire which was sent you, properly completed, together with your photograph.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The forty-third annual meeting of the Society was held at the Hotel Traymore, Atlantic City, June 3 to 5 inclusive, with 217 members and guests present. The after-remarks suggest that both the scientific and social programs were of exceptional merit.

During the business sessions several amendments to the Constitution and By-Laws were adopted. Eight candidates were elected to active Fellowship and two Fellows to Emeritus Fellowship.

The following officers were elected for the ensuing year: President, Samuel J. Kopetzky, New York. President-elect, Harold I. Lillie, Rochester, Minn. Vice-Presidents, Louis H. Clerf, Philadelphia; Murdock S. Equen, Atlanta; James B. Costen, St. Louis, and Arthur C. Jones, Boise. Treasurer, Kenneth M. Day, Pittsburgh. Editor, Lyman G. Richards, Boston. Secretary, C. Stewart Nash, Rochester, N. Y. Councillors: Class A—Perry G. Goldsmith, Toronto; Lee M. Hurd, New York, and Don M. Campbell, Detroit. Class B—Thomas E. Carmody, Denver; Harris P. Mosher, Boston, and Samuel Iglauer, Cincinnati. Class C—George M. Coates, Philadelphia; Ralph A. Fenton, Portland, Ore., and John J. Shea, Memphis. Ralph A. Fenton and Burt R. Shurly were elected representatives to the American Board of Otolaryngology for five years.

Since the Congress of American Physicians and Surgeons will meet in Atlantic City in May, 1938, and since the American Otological Society and the American Laryngological Association are constituent members of the Congress, it is expected that the Triological Society will adjust its schedule to meet that of the other Societies.

We announce with regret the retirement of Dr. Ewing W. Day from the office of Treasurer, which he held for 35 years. Untiring and uncompromising in his loyalty to the Society, he has always been its staunch guardian and trustee. The Society cannot let so ardent a supporter become inactive.